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CONTENTS OF VOLUME 11

July, 1927 CHICAGO NUMBER

Clinic of Dr. Lewis J. Pollock, *Northwestern University* PAGE

ANEURYSMS OF THE CEREBRAL VESSELS 1

Clinic of Dr. Francis Eugene Senear, *University of Illinois*

CUTANEOUS TUBERCULOSIS (SCROFULODERMA) AND TUBERCULIDS (ERYTHEMA INDURATUM, FOLLICLIS, ACNE SCROFULOSORUM) OCCURRING IN A CHILD WITH TUBERCULOSIS OF THE KNEE-JOINT 15

XANTHOMA TUBEROSUM MULTIPLEX OCCURRING IN A WOMAN WITH LATENT SYPHILIS 21

RECURRENT SECONDARY SYPHILIS OF THE SKIN AND MUCOUS MEMBRANES 25

BULLOUS IODODERMA. RAPID RECOVERY FOLLOWING THE USE OF INTRAVENOUS INJECTIONS OF SODIUM CHLORID 29

Clinic of Dr. Samuel J. Taub, *Cook County Hospital*

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS COMPLICATED WITH DIABETES MELLITUS 33

Clinic of Dr. Samuel M. Feinberg, *Cook County Hospital*

ASTHMA DUE TO HOUSE DUST 41

Clinic of Dr. Grant H. Laing, *St. Luke's Hospital*

MIGRAINE TYPES IN RELATION TO THE GASTRO-INTESTINAL TRACT 49

Clinic of Dr. E. F. Foley, *from the Research and Educational Hospitals, University of Illinois*

ALCOHOLIC CIRRHOSIS OF THE LIVER 55

Clinic of Dr. Harry A. Singer, *Research and Educational Hospital, University of Illinois College of Medicine*

WALLED-OFF AND FREE PERFORATION OF PEPTIC ULCER 63

Clinic of Dr. L. C. Gatewood, *Presbyterian Hospital*

DUODENAL ULCER COMPLICATED BY NEPHRITIS AND ALKALOSIS 79

Clinic of Dr. Robert W. Keeton, *Research and Educational Hospital, University of Illinois*

ESTIMATION OF THE SEVERITY OF THE DIABETIC DEFECT—TWO CASES 95

Clinic of Dr. George O. Solem, *Lutheran Deaconess Home and Hospital*

MASSIVE COLLAPSE OF THE LUNG 105

Clinic of Dr. James H. Hutton, *Chicago*

ENDOCRINE DISORDERS AS RELATED TO MORE COMMON SYNDROMES 113

Clinic of Dr. Isadore Pilot, *Research and Educational Hospital, University of Illinois*

FUSOSPIROCHETAL PULMONARY ABSCESS 127

Clinic of Drs. Charles A. Elliott, Walter H. Nadler, and Paul Starr, *Wesley Memorial Hospital, Northwestern University Medical School*

LIVER FEEDING IN PERNICIOUS ANEMIA 147

Clinic of Drs. Charles Spencer Williamson and Carroll L. Birch, *Research and Educational Hospital, University of Illinois*

A CASE OF ACUTE MYELOBLASTIC LEUKEMIA WITH ALEUKEMIC COURSE, WITH AUTOPSY FINDINGS 157

A CASE OF HEMOPHILIA, WITH GENEALOGICAL CHART 169

Clinic of Dr. Isaac A. Abt, *St. Luke's Hospital* PAGE
EXAMINATION OF THE INFANT. SPECIAL CONSIDERATION OF ABDOMINAL PALPATION..... 179

Clinic of Drs. Fred M. Drennan and Frank C. Val Dez, *Loyola University Medical School*
SIMPLE ULCERATIVE COLITIS..... 189

Clinic of Drs. Julius H. Hess and Joseph K. Calvin, *Michael Reese Hospital*
ALBUMINURIA IN CHILDREN..... 197

Clinic of Dr. Nathan Smith Davis, III, *Alexian Brothers Hospital*
CIRRHOSIS OF THE LIVER..... 215
CARCINOMA OF THE PANCREAS WITH METASTASES TO THE LIVER..... 219
CHRONIC INFECTIOUS JAUNDICE..... 221
ERYTHREMIA AND PODAGRA..... 225

Clinic of Dr. Arthur R. Elliott, *Medical Service, St. Luke's Hospital*
TYPHOID BONE INFECTION..... 229
PELLAGRA SECONDARY TO CARCINOMA OF THE COLON..... 237

Clinic of Dr. Jacob Meyer, *Michael Reese and Cook County Hospitals*
GASTRIC ULCER HEMORRHAGE AND ITS TREATMENT..... 245
ACUTE NEPHRITIS—TREATMENT..... 249

Clinic of Dr. James G. Carr, *Cook County Hospital*
AMEBIC DYSENTERY..... 255

Clinic of Dr. Jesse R. Gerstley, *Department of Pediatrics, Northwestern University Medical School and Michael Reese Hospital*
OUR BABY PATIENT..... 269

Clinic of Dr. Emmet B. Bay, *Presbyterian Hospital*
UNUSUAL PROGRESS IN HEART IRREGULARITIES..... 279

Clinic of Drs. Charles Louis Mix and John F. Golden, *Mercy Hospital*
OBSTRUCTION OF THE DUODENUM BY BANDS OF ADHESIONS..... 287

September, 1927 ST. LOUIS NUMBER

Contribution by Drs. Elsworth S. Smith and Hiram S. Liggett, *from the Department of Internal Medicine, Washington University School of Medicine* PAGE
FURTHER CLINICAL STUDIES IN ESSENTIAL ARTERIAL HYPERTENSION..... 295

Clinic of Dr. John Zahorsky, *St. Mary's Hospital*
GIARDIASIS IN AN INFANT..... 311
INFECTION BY THE TRICHOMONAS HOMINIS..... 315
INFANTILE ECLAMPSIA..... 317
EXUDATIVE ENTERITIS..... 319

Clinic of Dr. Andrew B. Jones, *Washington University School of Medicine*
A FAMILY OF FAMILIAL CLAW-FOOT..... 325
DYSTROPHIA MUSCULORUM PROGRESSIVA..... 339
THOMSEN'S DISEASE (MYOTONIA CONGENITA)..... 341
FRIEDREICH'S DISEASE (HEREDITARY ATAXIA)..... 343

Clinic of Dr. Ralph A. Kinsella, *St. Mary's Hospital (University Hospital, St. Louis University School of Medicine)*
THE CLINICAL IMPORTANCE OF THE RELATION BETWEEN THE CONNECTIVE TISSUE OF THE STOMACH, DUODENUM, PANCREAS, LIVER, AND SPLEEN..... 345

Clinic of Dr. Albert E. Taussig, *from the Medical Service of the Jewish Hospital*
PURE CONGENITAL DEXTROCARDIA..... 351
RENAL GLYCOSURIA WITH KETONURIA..... 355

Clinic of Dr. Sidney I. Schwab, *Barnes Hospital (Washington University)*
PSYCHIATRIC SYMPTOMS IN MEDICAL OR SURGICAL DISEASES..... 361

PAGE

Clinic of Dr. William Engelbach, *Jewish Hospital*
INFANTILE DEFECTIVENESS 1

Clinic of Dr. Harry L. Alexander, *from the Department of Internal Medicine, Washington University School of Medicine*
LOCALIZED ALLERGY 393

Clinic of Dr. Horace W. Soper, *St. Luke's Hospital*
TREATMENT OF PEPTIC ULCER 405

Clinic of Dr. Borden S. Veeder, *Washington University School of Medicine*
THE SYSTEMIC MANIFESTATIONS OF UPPER RESPIRATORY INFECTIONS 415

Clinic of Dr. Francis M. Barnes, Jr., *St. Louis City Sanitarium*
EPILEPTIC MENTAL DISORDERS 425

Clinic of Dr. Goronwy O. Broun, *from the Department of Medicine, St. Louis University School of Medicine, and the Medical Service of St. Mary's Infirmary*
A CASE OF PERNICIOUS ANEMIA WITH PRONOUNCED NEUROLOGIC SYMPTOMS, WITH REMARKS ON CERTAIN BACTERIOLOGIC AND SEROLOGIC FEATURES OF THE DISEASE 439

Clinic of Dr. Drew Luten, *Barnes Hospital*
CORONARY THROMBOSIS OF LESSER SEVERITY WITH SPECIAL REFERENCE TO THE VALUE OF ELECTROCARDIOGRAPHY IN THE DIAGNOSIS 445

Clinic of Dr. David P. Barr, *Barnes Hospital*
THE DIAGNOSIS OF MULTIPLE LIVER ABSCESS 455

Clinic of Dr. Walter Baumgarten, *from the Medical Service, St. Luke's Hospital*
THE RELATIONSHIP BETWEEN CHOLECYSTITIS AND THE PERSISTENCE OF DUODENAL ULCER 473

Clinic of Dr. J. Curtis Lyter, *St. Anthony's Hospital*
CHYLOUS ASCITES AND CHYLOTHORAX 479

Clinic of Dr. Arthur E. Strauss, *from the Heart Clinic of Washington University Dispensary, the Barnes Hospital, and the Department of Internal Medicine of Washington University*
HEART-BLOCK, AURICULAR FLUTTER, AND ADENOMA OF THE THYROID 487

Clinic of Dr. Joseph W. Larimore, *Barnes Hospital (Washington University Medical School)*
PHYTOBEZOAR 499

Clinic of Dr. Joseph Grindon, *St. Louis University School of Medicine*
NOTES ON THE DAVIS TREATMENT AND TRYPARSAMID IN ACUTE PEMPHIGUS 507

Clinic of Dr. Alphonse McMahon, *St. John's Hospital*
THE ASSOCIATED CARDIAC STATES IN HYPERTHYROIDISM 515

Clinic of Dr. James F. McFadden, *St. John's Hospital*
BRADYAPNEA OR TRANSITORY BILATERAL VAGUS PARALYSIS IN TABES DORSALIS 531
JUVENILE PARESIS 537
NEUROPSYCHIATRIC MANIFESTATIONS OF BROMISM 541
EMBOLIC HEMIPLEGIA COMPLICATED BY EMBOLISM OF PERIPHERAL ARTERIES 547

Clinic of Dr. O. P. J. Falk, *St. John's Hospital*
THE DIET PROBLEM IN CHRONIC BILE-TRACT DISEASE 553

Clinic of Dr. A. R. Shreffler, *Deaconess Hospital*
DIAPHRAGMATIC HERNIA 561

Clinic of Dr. Samuel B. Grant, *Missouri Baptist Sanitarium*
THE MANAGEMENT OF GOITER PATIENTS WITH CONGESTIVE HEART-FAILURE 569

Clinic of Dr. Joseph P. Costello, *St. John's Hospital*
THYMIC ENLARGEMENT AS A CAUSE OF ATELECTASIS 583

Clinic of Dr. Charles Hugh Neilson, *St. Louis University*
THE NERVOUS BREAKDOWN 591

Clinic of Dr. Daniel L. Sexton, *Jewish Hospital*
THE DIAGNOSTIC VALUE OF BLOOD CALCIUM ESTIMATION 603

November, 1927 TULANE UNIVERSITY NUMBER

PAGE

Clinic of Dr. John H. Musser, *Tulane University*
MEASLES ... 619

Clinic of Dr. Charles W. Duval, *Tulane University*
SCARLET FEVER ... 631

Clinic of Dr. I. I. Lemann, *Tulane University*
THE TREATMENT OF DIABETIC GANGRENE. PROPHYLAXIS. WHEN TO AMPUTATE. WHEN TO PRACTICE CONSERVATISM ... 641

Clinic of Dr. Oscar W. Bethea, *Tulane University*
PLEURISY ... 653

Clinic of Drs. Ralph Hopkins and O. E. Denney, *United States Marine Hospital No. 66.*
A PHOTOGRAPHIC DEMONSTRATION OF SOME OF THE MACULAR LESIONS OF LEPROSY ... 667

Clinic of Dr. Henry Daspit, *Tulane University*
TWO INTERESTING SKULL TUMORS ENCROACHING ON THE INTRACRANIAL SPACE ... 675

Clinic of Dr. Allan C. Eustis, *Baptist Hospital*
INDIVIDUALIZATION IN THE TREATMENT OF DISEASE ... 687

Clinic of Dr. Chaillé Jamison, *Tulane University*
STREPTOCOCCUS PNEUMONIA ... 697

Clinic of Dr. C. L. Eshleman, *Touro Infirmary*
PURPURA HÆMORRHAGICA ... 701

Clinic of Dr. George R. Herrmann, *Tulane University and Charity Hospital*
PURPURA AS A CARDIOLOGIC PROBLEM ... 719

Clinic of Dr. James M. Bamber, *Tulane University*
SARCOMA OF THE STOMACH ... 753
FOREIGN BODY WITHIN THE ABDOMEN ... 765
EVENTRATION OF THE DIAPHRAGM ... 771

Clinic of Dr. F. M. Johns, *Tulane University*
INTERESTING CLINICAL LABORATORY FINDINGS IN CHRONIC PULMONARY ACTINOMYCOSIS AND THE AMERICAN VARIETY OF CUTANEOUS LEISHMANIASIS ... 775

Clinic of Dr. R. W. Mendelson, *United States Marine Hospital, New Orleans*
PLAGUE ... 779

Clinic of Dr. Daniel N. Silverman, *Touro Infirmary*
A CLINICAL METHOD FOR THE QUANTITATIVE DETERMINATION OF STOOL REACTION ... 801

Clinic of Dr. Sam Hobson, *Tulane University*
A BLOOD-DYSCRASIA-DIARRHEA CASE PRESENTED AS A DIAGNOSTIC PROBLEM ... 805

Clinic of Dr. Leon J. Menville, *Tulane University*
THE APPLICATION OF MODERN METHODS IN THE STUDY OF THE VISUALIZED GALL-BLADDER BY MEANS OF THE ROENTGEN-RAYS ... 811

January, 1928 BROOKLYN NUMBER

PAGE

Clinic of Dr. Henry Monroe Moses, *Kings County Hospital*
PROBLEMS IN DIAGNOSIS ... 831

Clinic of Dr. Henry Joachim, *Long Island College Hospital*
TWO CASES OF ASCITES ... 851

Clinic of Dr. Irving L. Cabot, *Methodist Episcopal Hospital*
USE OF PHENYLHYDRAZIN IN CASE OF POLYCYTHEMIA COMPLICATED BY DIABETES ... 863

Clinic of Dr. Archibald D. Smith, *From the Pediatric Service of the Brooklyn Hospital*
INTRACRANIAL HEMORRHAGE OF NEWBORN. REPORT OF SIXTEEN CASES WITH THREE AUTOPSIES ... 869

Clinic of Dr. Edward E. Cornwall, *Norwegian Hospital* PAGE
MANAGEMENT OF THE ALIMENTARY TRACT IN PNEUMONIA, WITH SPECIAL REFERENCE TO THE DIET AND THE ARTIFICIAL INDUCTION OF BOWEL EVACUATIONS 879

Clinic of Dr. Irving Gray, *Jewish Hospital*
GASTRIC SYMPTOMS OCCURRING IN SYPHILITICS 889

Clinic of Dr. Benjamin Kramer, *From the Pediatric Department and the Pathological Laboratory of the Jewish Hospital*
LIPOID-CELL SPLENOHEPATOMEGALY, NIEMANN-PICK TYPE. A CLINICAL ANALYSIS OF NINE CASES WITH A REPORT OF ONE ADDITIONAL CASE 905

Clinic of Dr. Tasker Howard, *Long Island College Hospital*
COMPENSATION AND REPAIR. SOME CLINICAL EVIDENCE BEARING ON OPTIMISM IN DIAGNOSIS 919

Clinic of Dr. A. L. Louria, *Jewish Hospital*
SYPHILIS OF THE LUNG SIMULATING TUBERCULOSIS OR CARCINOMA OF THE LUNG 931
SYPHILIS OF THE CENTRAL NERVOUS SYSTEM SIMULATING DIABETES 937

Clinic of Dr. Charles Shookhoff, *Jewish Hospital*
THE TACHYCARDIAS 941

Clinic of Dr. M. C. Myerson, *Jewish Hospital*
BRONCHOSCOPY IN THE TREATMENT OF LUNG SUPPURATION 961

Clinic of Drs. Morris M. Banowitch and Gordon H. Ira, *Long Island College Hospital*
EMBOLISM AND THROMBOSIS OF THE ABDOMINAL AORTA. REPORT OF FIVE CASES 973

Clinic of Dr. Albert F. R. Andresen, *From the Gastro-enterological Department, Long Island College Hospital*
CARCINOMA OF THE ESOPHAGUS. WITH REPORT OF FOUR UNUSUAL CASES 1001

Clinic of Dr. Meyer A. Rabinowitz, *Jewish Hospital*
TOXIC HEPATITIS AND HEPATOLYSIS FOLLOWING THE USE OF ATOPHAN 1025

Clinic of Dr. John N. Evans, *Brooklyn Hospital*
INTERPRETATION OF EYE PATHOLOGY. CLINICAL PERIMETRY—ITS VALUE TO THE INTERNIST 1043

Clinic of Dr. Harry M. Greenwald, *United Israel-Zion Hospital*
DYSTROPHY IN ARTIFICIALLY FED INFANTS 1055

Clinic of Dr. Henry M. Feinblatt, *Long Island College Hospital*
THREE CASES OF ENDOCRINE DISEASE WITH PRONOUNCED ASTHENIA 1071

Clinic of Dr. Frank Bethel Cross, *Methodist Episcopal Hospital*
THE CLINICAL TYPES OF CARDIAC FAILURE 1081

Clinic of Dr. S. R. Blatteis, *Jewish Hospital*
ENDEMIC TYPHUS FEVER (BRILL'S DISEASE). A REPORT OF 138 CASES 1099

March, 1928 TULANE UNIVERSITY NUMBER

Clinic of Dr. C. C. Bass, *Tulane University* PAGE
MALARIAL HEMOGLOBINURIA 1109
HEALTH EXAMINATION 1117

Clinic of Dr. Aldo Castellani, *Tulane University and Charity Hospital*
INTERNAL MYCOSES 1123

Clinic of Dr. J. Holmes Smith, Jr., *Tulane University*
SICKLE-CELL ANEMIA 1171

Clinic of Dr. Elizabeth Bass, *Tulane University*
THE LEUKOCYTES 1191

Clinic of Dr. Roy H. Turner, *Tulane University and Charity Hospital*
THE VALUE OF THE OBSTETRIC HISTORY IN MAKING A DIAGNOSIS OF SYPHILIS 1211

Clinic of Dr. H. W. Butler, *Tulane University* PAGE
THE RESULTS OF ONE COURSE OF TREATMENT WITH OIL OF CHENOPODIUM ON A GROUP OF PATIENTS HEAVILY INFESTED WITH INTESTINAL PARASITIC WORMS 1221
SYPHILIS AND THE SLIDE TEST 1225

Clinic of Dr. Russell C. Pigford, *Tulane University*
THE RELATIONSHIP OF CRYPTORCHIDISM TO ENDOCRINOLOGY 1231

Clinic of Dr. Willard R. Wirth, *Tulane University*
THE VALUE OF RED CELL DIAMETER ESTIMATIONS IN ANEMIAS 1247

Clinic of Dr. L. E. Williford, *Tulane University*
TREATMENT OF PARESIS BY MALARIA, SODAKU, AND TRYPARSAMID 1269

Clinic of Dr. Dudley M. Stewart, *Tulane University*
HYPERTROPHIC OSTEO-ARTHROPATHY 1283

Clinic of Dr. R. F. Sowell, *Charity Hospital*
THE EFFECTS OF ANTITOXIN ON THE LEUKOCYTOSIS IN DIPHTHERIA 1293

Clinic of Dr. D. R. Womack. *Charity Hospital*
MENINGOCOCCAL MENINGITIS TREATED WITH CISTERNAL PUNCTURE 1299

Clinic of Dr. C. McD. Smith, *Tulane University*
A CASE OF ECHINOCOCCUS CYST OF THE LIVER AND THE PERITONEAL CAVITY 1313

Clinic of Dr. Edgar Hull, *Tulane University*
ATYPICAL TUBERCULOUS ADENITIS COMPLICATED BY MULTIPLE TUMORS OF THE BONE-MARROW. REPORT OF A CASE 1321

Clinic of Dr. A. N. Houston, *Tulane University*
ANALYSIS OF EIGHTY-EIGHT CASES OF RHEUMATIC FEVER. COMPARISON WITH OTHER ANALYSES AND DISCUSSION 1339

Clinic of Dr. M. Mallowitz, *Tulane University*
THE RESIDUALS OF EPIDEMIC ENCEPHALITIS 1351

Clinic of Dr. C. D. Overton, *Tulane University*
DIVERTICULOSIS OF THE LARGE BOWEL 1361

May, 1928 MAYO CLINIC NUMBER

Cases Discussed in the Clinics of Dr. George B. Eusterman PAGE
RECURRENT GASTRIC ULCER AND ASSOCIATED GASTROJEJUNAL ULCER IN THE PRESENCE OF CONSTANT GASTRIC ANACIDITY 1371
HEMOCHROMATOSIS 1376

Howard R. Hartman
A STUDY OF THE CAUSE OF JAUNDICE IN FOUR HUNDRED CASES 1383

Porter P. Vinson and Herman J. Moersch
THE DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE LOWER PART OF THE ESOPHAGUS AND CARDIAC END OF THE STOMACH 1389

Alexander B. Moore
CERTAIN PRACTICAL POINTS IN THE ROENTGENOLOGIC DIAGNOSIS OF GASTRIC ULCER ... 1399

James F. Weir
THE PREOPERATIVE TREATMENT OF COMPLICATIONS OF GASTRODUODENAL DISEASE 1407

Charles S. McVicar
JAUNDICE ASSOCIATED WITH PERIPHERAL NEURITIS 1415

Albert M. Snell
PROBLEMS IN THE TREATMENT OF PATIENTS WITH ASCITES 1419

Walter C. Alvarez
HYPERTENSION AND APPENDICITIS IN EIGHT MEMBERS OF ONE FAMILY 1435

PAGE

Philip W. Brown
CERTAIN ASPECTS OF ENDAMEBIASIS 1449

Cases from the Section of Leda J. Stacy
PRECOCIOUS PUBERTY (LEDA J. STACY) 1455
ANOMALIES OF THE FEMALE GENERATIVE TRACT (L. MARY MOENCH) 1457
HYPERTROPHY OF THE BREASTS (SUSAN R. OFFUTT) 1459
ADENOMYOMA OF THE UTERUS IN YOUNG WOMEN (DELLA G. DRIPS) 1460
PROLAPSE OF UTERUS IN A YOUNG WOMAN (DELLA G. DRIPS) 1462

Robert D. Mussey
PREÉCLAMPTIC TOXEMIA 1463

Lawrence M. Randall
OCCIPUT-POSTERIOR PRESENTATION 1469

Benjamin H. Hager and William F. Braasch
UNUSUAL UROLOGIC CASES 1479

Leonard G. Rowntree
SOME SIDELIGHTS ON HYPERTENSION 1509

Edgar V. Allen
CLINICALLY ARRESTED THROMBO-ANGIITIS OBLITERANS 1527

Bayard T. Horton
HYPERTENSION AND POLYCYTHEMIA: THE SO-CALLED GEISBOCK'S SYNDROME 1535

George R. Constam and George E. Brown
CYANOSIS 1543

Walter R. Johnson
RECURRENT IDIOPATHIC SUPERFICIAL PHLEBITIS: ITS SIGNIFICANCE IN ADULT MEN 1559

Edwin G. Bannick and Norman M. Keith
ACUTE RENAL INSUFFICIENCY FOLLOWING SURGICAL OPERATION FOR CARCINOMA OF THE SIGMOID AND RECTUM 1571

Harry L. Parker
RECURRENT ATTACKS OF UNCONSCIOUSNESS 1583

Charles G. Sutherland
OSTEITIS INVOLVING THE BONES OF THE PELVIS 1593

Philip S. Hench and Harry Fortin
THE CARE OF THE FEET IN CHRONIC ARTHRITIS 1603

Edwin B. Rentschler
"GOUTY ARTHRITIS": THE IMPORTANCE OF PERIODICITY WITH COMPLETE REMISSIONS AS AN AID IN EARLY DIAGNOSIS 1627

Paul A. O'Leary
SYPHILIS OF THE CENTRAL NERVOUS AND CARDIOVASCULAR SYSTEMS IN HUSBAND AND WIFE 1643

Frank N. Allan and Russell M. Wilder
THE TREATMENT OF ERYSIPELAS WITH ANTITOXIN 1653

Thomas B. Magath
UNDULANT FEVER. THREE CASES 1661

Lee W. Pollock and Clifford J. Barborka
ABDOMINAL MIGRAINE; MELANOMA; PELLAGRA; HEPATOLENTICULAR DEGENERATION (WILSON'S DISEASE) 1665

Index to Volume 11 1673

THE MEDICAL CLINICS
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Volume 11 No. 1

CLINIC OF DR. LEWIS J. POLLOCK

NORTHWESTERN UNIVERSITY

ANEURYSMS OF THE CEREBRAL VESSELS

ALTHOUGH of rare occurrence, cerebral aneurysms frequently present difficult problems in differential diagnosis. The correct diagnosis of cerebral aneurysm during the life of the patient is exceedingly difficult and the pathologist has much more experience with the disease than the clinician. Symonds has published the records of 5 cases in which he made the diagnosis of cerebral aneurysm during life, and was able later to confirm his diagnosis at necropsy in three instances. In a material of 5432 necropsies on the head Fernsides was able to collect 44 cases of cerebral aneurysm.

In this clinic only true fusiform or saccular aneurysms which are easily visible to the naked eye will be considered. We will not refer to the miliary aneurysms which have attributed to them a problematical importance in the occurrence of cerebral hemorrhage. Arteriosclerosis, or atheromatous degeneration of the media, may be considered as one of the more common causes of large aneurysms, but there are many cases in which there is no evidence of arteriosclerosis anywhere in the body, and the patients often are quite young. Developmental defects about the circle of Willis, particularly at the junction of the anterior cerebral and communicating arteries, play an important rôle in the production of the aneurysms in young individuals. General infection, infective endocarditis, and septic emboli play a

not unimportant part. Syphilis seems to have a very small part in the formation of intracranial aneurysms.

The symptoms and signs produced by cerebral aneurysm are dependent upon the location and size of the aneurysm and the condition of the aneurysmal sac. An unruptured cerebral aneurysm may produce the general signs and symptoms of increased intracranial tension and focal signs dependent upon the location of the aneurysm. Occasionally such cases have been interpreted as cerebral neoplasms and have been operated upon.

Aneurysms of the internal carotid and middle cerebral arteries may produce compression of the ophthalmic division of the fifth nerve, paralysis of the third, fourth, and sixth cranial nerves, and occasionally papilledema. At times the other branches of the fifth nerve may likewise be involved.

An instructive case may be cited. A woman aged sixty-five was admitted to the service of Dr. A. B. Kanavel, at Wesley Memorial Hospital, on October 9, 1919. She gave a history of having had attacks of pain beginning in February, 1919, lasting over a period of six weeks. The pains were paroxysmal, lightning-like, short, momentary, starting from a point over the mental foramen of the left lower jaw, radiating upward, and were brought on by talking or rubbing the tongue against the alveolar process. They were never produced by stimulation of any other algigenetic zones. These pains disappeared after six weeks and did not reappear until six days prior to her admittance to the hospital, when she again experienced similar paroxysms and, in addition, each attack was associated with a sharp shoot of pain in the left eyeball. Rubbing the skin in front of the left ear now produced pain in the lower jaw. She complained of aching and a bulging feeling in the left eyeball.

The subjective symptoms of paroxysmal pains of short duration brought on by eating, talking, and rubbing an algigenetic or trigger zone were typical of a trifacial neuralgia. Pains in the distribution of the fifth nerve may be due to many causes, and in a clinic where patients suffering from trifacial neuralgia are treated by alcohol injections or other surgical procedures it is common to find instances of face pain due to cerebral tumors,

tabes dorsalis, syringobulbia, syphilitic meningitis, diseases of the paranasal sinuses, and diseases of the jaws and teeth. In none of these instances, however, does the pain conform to the description which we have already given. Long lasting pains not produced by stimulating any trigger zone are the rule. It is common for such patients to freely point to and handle that part of the face where the pain appears, in contrast to a patient suffering from trigeminal neuralgia, who will not permit an examiner to come within several feet of him, holding his hands and head in a defensive attitude.

In the case we are describing the diagnosis of trigeminal neuralgia had been made and, prior to a contemplated operation, a neurologic examination was being conducted. It was found that there was no paralysis or paresis of any of the extremities or motorcranial nerves. The deep reflexes were normal, as were the superficials, with the exception of the abdominals, which were absent because of marked adiposity. There was no change in tone, synergy, or co-ordination. There was middle-ear deafness on the left side. There was no objective sensory loss over the extremities, tongue, or face. The right pupil was smaller than the left; both were slightly irregular. The right reacted promptly to light, whereas the left reacted sluggishly.

A note was made that the history and course of the illness was compatible with typical trifacial neuralgia, but that because of pupillary inequalities operation should be delayed to determine the reason for this condition.

Upon the following day the patient confirmed the history of attacks of pain starting in the lower jaw, paroxysmal and momentary in duration, and brought on by rubbing the side of the tongue against the cheeks and gums. No headaches or vomiting were complained of, but on that day she experienced some dizziness. The deep reflexes were slightly diminished. No pathologic reflexes were found, there was no paralysis or paresis, no cerebellar disturbance, and no sensory disturbance even in the distribution of the fifth nerve. The motor distribution of the fifth nerve seemed normal. Early in the morning

she noted that she was unable to open the left eye, and examination revealed a paralysis of the third nerve, with ptosis, marked dilatation of the pupil, paralysis of the internal, superior, inferior rectus, and a paralysis of the fourth nerve, the only movement possible being that of external rotation. Taste was slightly diminished on the left side of the tongue.

Ophthalmoscopic examination showed the veins of the left disk to be engorged and the borders of the disk somewhat indistinct. On the right side the veins were engorged and but little cupping was observed.

The opinion was expressed that there was possibly a tumor in the region of the gasserian ganglion, such as have been described as tumors of the paratrigeminal area, usually originating from the sympathetic nervous system.

The next day the patient showed slight clouding of consciousness. On October 15th she lost consciousness, had marked stertorous breathing, the head and eyes were turned to the left, there was a bilateral Babinski, but no Kernig or Brudzinski. On October 16th the general condition was very poor, she was deeply unconscious, the temperature had risen to 104° F., the eyes showed a conjugate deviation to the left, and the bilateral Babinski persisted. Ophthalmoscopic examination showed no further change than that recorded. The respirations became very rapid, ranging from 48 to 58, and upon one occasion the pulse was slow—66.

A spinal puncture was performed on October 14th, prior to the development of the unconsciousness, and did not reveal the presence of blood, but showed a slight increase in protein content, with a negative Wassermann reaction on both spinal fluid and blood.

The patient died on October 16th, and necropsy revealed a diffuse subarachnoid hemorrhage at the base of a small saccular aneurysm, about the size of a large pea, at the junction of the left middle cerebral and posterior communicating arteries.

The case brilliantly illustrates the difficulties in differential diagnosis. First, attention must be called to the great similarity of the symptoms to classical trigeminal neuralgia. Had there

not been the slight inequality of the pupils this patient would have been operated on for section of the sensory root of the gasserian ganglion. Fortuitously, bleeding from the aneurysm began at this time and produced the symptoms which pointed to a definite focal lesion about the paratrigeminal area. The signs and symptoms then and until her death were quite compatible with a tumor in this locality.

Aneurysms in the anterior group of vessels of the circle of Willis, therefore, rather regularly produce symptoms referable to the third, fourth, fifth, and sixth cranial nerves.

In addition to the focal signs produced by the compression of an aneurysm, leakage from an aneurysm from time to time may produce a train of symptoms which are exceedingly suggestive, and which upon rare occasions permit of a diagnosis before death.

A woman aged forty-two was admitted to the Michael Reese Hospital on November 10, 1926, with the following history: She has been perfectly well until November 10, 1926, when, after making a telephone call, she sat down, suddenly felt dizzy, and developed a terrific pain in her head and projectile vomiting. The pain was so severe as to cause her to scream out in agony. Shortly afterward she had a clonic convulsion of the arms, but did not lose consciousness.

The history of her previous status revealed the fact that about a year before she had fainted after getting out of a bath, and had a headache which lasted about five minutes. During the preceding year she had complained of some aching in her eyeballs.

Shortly after her admission to the hospital she was found lying in bed, screaming with pain. There was an external strabismus of the left eyeball, which was rotated slightly upward and outward. There seemed to be slight weakness in the left lower extremity. The deep reflexes of the lower extremities were lost, the wrist-jerk was diminished on the right, less so on the left. The ulnar jerks were diminished, the left being greater than the right. The biceps-jerks were absent, the triceps-jerks were normal. The right upper eyelid did not completely

close. There was a suspicion of a Babinski on the right side. On the left side plantar stimulation produced dorsal flexion of all toes. The right lower abdominal reflex was present, the left was diminished. The upper abdominal reflexes were absent. There was no contralateral Brudzinski or Kernig. There was marked variation of pulse pressure, many beats failing to come through at the upper limit of systolic pressure. There was marked rigidity of the neck, and upon anteflexion of the head the right arm and leg flexed more frequently than did the left. The heart borders were slightly enlarged beyond the normal limits, the heart beat was very irregular.

In considering the differential diagnosis an impression was noted of the possibility of a brain tumor, cerebral embolism consequent to auricular fibrillation, or subarachnoid hemorrhage.

Upon the following day ophthalmoscopic examination did not reveal a choked disk, or were other signs of increased intracranial pressure definitely noted, with the exception of continuation of the rigidity of the neck. The left Achilles-jerk was absent, the right knee-jerk was greater than the left, as was also the right wrist-jerk. There was no objective sensory loss; there was a very slight Kernig sign on both sides.

It was felt that a spinal puncture could safely be made, and 10 c.c. of bloody fluid were obtained under 10 mm. of mercurial pressure. It was at first thought possible that the blood was due to trauma. Differential blood-count on the spinal fluid did not show a pleocytosis. There was an increase in non-protein nitrogen in the blood and a slight trace of albumin in the urine.

Upon the following day the patient was perfectly conscious and was able to give some additional history of having had rather severe headache several times a week for a considerable number of months. They were usually occipital, varying in character, sometimes dull, sometimes sharp. A feeling of dizziness usually accompanied the headache, and the headache usually lasted for but a few moments.

On November 13th there was increase in the rigidity of the neck, the temperature rose to 100.4° F., and she had a leukocytosis of 17,000. On November 15th she complained of slight

numbness in the right arm. On November 18th she suddenly lost consciousness, had Cheyne-Stokes respiration, the left leg and arm usually were held rigidly, the leg extended, the arm flexed. She moved the right arm upon painful stimulation, but not the left. The right foot was withdrawn when the sole was pricked with a needle, the left was not. There was a conjugate deviation of the eyes to the right. Rotation of the chin to the right was followed by flexion of the left leg, and rotating the chin to the left produced flexion of the right leg. The deep reflexes seemed slightly greater on the right side.

Another spinal puncture at this time showed a diffusely bloodly spinal fluid, and it was concluded that the blood was the result of subarachnoid hemorrhage.

The marked rigidity of the neck, the presence of Magnus de Klejn reflexes upon turning the head from side to side, with ensuing flexion of the lower extremities, all pointed to a lesion at the base of the brain, and the bloody spinal fluid indicated a subarachnoid hemorrhage. Although spontaneous subarachnoid hemorrhages are commonly encountered in the newborn, it is my experience that in adults this condition rarely occurs except when some underlying pathology, such as a chronic meningitis, arteriosclerosis, or other vascular lesions, dementia paralytica, chronic alcoholism, uremia, diabetic coma, toxic states of pregnancy, etc., are present.

The history of long-continued headaches, an attack of loss of consciousness a year before in an otherwise healthy individual, followed by an attack characterized by very severe, excruciating pain in the head, projectile vomiting, and blood in the spinal fluid, this followed by a marked amelioration of signs and symptoms, and this, in turn, followed by another attack with complete loss of consciousness and increase of signs of subarachnoid hemorrhage, pointed to recurrent subarachnoid hemorrhage, which would most probably be the result of aneurysm of the cerebral vessels, and this diagnosis was made prior to the patient's death, on November 18th.

Necropsy showed an extensive subarachnoid hemorrhage over the entire base of the brain, thickly matted over the pons

and peduncles. Bright red, fresh blood was present over the vertex in the subdural space, probably having perforated the arachnoid. At the junction of the anterior carotid and middle cerebral artery there was a small aneurysm, 2 x 1½ cm., partially thrombosed and calcified, which had ruptured. The temporal lobe was engorded at this point by a large blood-clot. Blood had penetrated all the sulci at the base, but other brain destruction was not noted. Blood was present in all the ventricles, having seeped in through the fourth ventricle. There was no evidence of any vascular disease other than at the point of the aneurysm.

Although fewer focal signs were present in this case, the diagnosis was not so difficult because of the suggestive train of symptoms of repeated attacks of extravasation of blood at the base of the brain. Such attacks are characterized by disturbance of consciousness, slight pyrexia, marked pain in the head, and evidence of meningeal irritation, perhaps here represented by the marked rigidity of the neck. The Magnus de Klejn reflexes are indicative of an interruption of fibers at the brain stem, and occur as a part of the picture of partial decerebration.

The original impression of the possibility of cerebral embolus was unconfirmed because of the lack of focal signs when the patient regained consciuosness, and the presence of bloody spinal fluid. No evidence of meningitis could be seen in the differential cell count of the spinal fluid. A cerebral neoplasm was ruled out because of the absence of focal signs, and the absence of any signs of increased intracranial tension, including the low spinal fluid pressure. There was no evidence of any systemic disease which would be likely to produce a spontaneous subarachnoid hemorrhage. The Wassermann reaction on both blood and spinal fluid was negative, thus ruling out a dementia paralytica with a pachymeningitis hæmorrhagica.

Although no notes of aneurysm of the posterior group are available in my material, it must be pointed out that the clinical picture which is produced by aneurysm of the basilar or vertebral arteries is quite definite, inasmuch as signs of pressure on the adjacent brain stem are usually quite prominent, with-

out evidence of paroxysmal leakage from the aneurysm. Recurrent paralyses, at times paraplegia, cerebellar symptoms, dysarthria and dysphagia, as well as contralateral hemanesthesia and paralysis of the last four cranial nerves are common.

THROMBOSIS OF BASILAR ARTERY

The symptoms of aneurysm of the posterior group of basilar vessels, particularly if due to slow hemorrhage, frequently are indistinguishable from those due to thrombi of the basilar artery, which is illustrated by the following case:

A man aged thirty-two was admitted to the Chicago Memorial Hospital on November 16, 1923. His family and personal history was of little interest in relation to his condition. He had suffered from the usual diseases of childhood, and at the age of twenty developed what was called a chancroid ulcer on the penis. No further symptoms of syphilis appeared and he received no treatment other than local.

Two weeks prior to his admission to the hospital he experienced a sudden vertiginous attack, with objective vertigo to the right. At the same time he was nauseated and vomited. A few hours later he experienced some difficulty in walking because of unsteadiness, and developed a headache of moderate severity. He remained in bed upon the following day, complaining of headache, dizziness, and a moderate degree of somnolence. These symptoms continued for two or three days, when they subsided, and he was able to resume his occupation.

Three days prior to admission to the hospital he had another attack of vertigo, associated with very marked vomiting and severe headache, and he fell to the floor as the result of partial loss of consciousness. During the succeeding period prior to his admission to the hospital he remained in a semiconscious state, at times complaining of headache, and it was noted that he was unable to swallow liquids without regurgitation or coughing, that some difficulty was experienced in swallowing solids, and that he was unable to use his right upper extremity with any degree of precision or strength.

Upon examination at the hospital it was found that there

was a moderate rigidity of the neck, and upon anteflexion of the head both arms and legs flexed, constituting a Brudzinski sign. There was no Kernig. When the upper extremities were extended forward and he was requested to hold them in that position with the eyes closed, the right gradually sank to the bed. Movements of the right hand were performed slowly and clumsily, and the strength in the right upper extremity was diminished. The right lower extremity when passively flexed and relaxed fell outward, but the left lower extremity remained in the position which was passively imposed upon it. In the left upper extremity evidences of cerebellar disturbance were found, consisting of inability to check movements promptly, inability to repeat such movements as pronation and supination, and in the presence of a rather gross tremor, which increased upon intentional movements.

There was a moderate loss of sensation over the right side of the body and the left side of the face. There was paresis of the left side of the palate and of the left superior constrictor of the pharynx. The tongue was protruded to the left, and when allowed to rest in the floor of the mouth it was pulled transversely to the right, indicating a paresis of the left side of the tongue. His voice was slightly hoarse, and a paresis of the left vocal cord was observed upon laryngoscopic examination.

The deep reflexes were moderately increased upon the right side and a right Babinski was present. Ophthalmoscopic examination showed normal disks. Spinal fluid was obtained under normal pressure. It was clear, showed 16 red cells per cubic millimeter, and a slight increase in protein content. The Wassermann reaction upon the blood and spinal fluid was negative.

The differential diagnosis rested between a tumor of the brain stem, thrombosis of the posterior inferior cerebellar artery or other branches of the basilar artery, and an aneurysm of the posterior group of basilar cerebral vessels.

The absence of evidence of increased intracranial tension, such as slow pulse, high blood-pressure, respiratory disturbance, choked disk, long-lasting headaches and vomiting, served to exclude a brain tumor. The posterior inferior cerebellar artery

is the seat of election for thrombotic formations in the basilar vessels of the posterior fossa. In such a case the symptomatology is typical, limited to ipsolateral cerebellar disturbance, analgesia over the fifth nerve, paralysis of the cervical sympathetics distributed by the fifth nerve, and a contralateral hemianalgesia. In this particular case, in addition to most of these findings, there was an ipsolateral involvement of the ninth, tenth, eleventh, and twelfth cranial nerves, and a contralateral hemiparesis.

It was, therefore, apparent that an area larger than that which is supplied by the posterior inferior cerebellar artery was involved in the pathology. Apoplectiform bulbar palsies usually occur with a more sudden onset, are bilateral, and associated with bilateral spastic paresis. They are usually due to small focal lesions and the acute attack is ordinarily recovered from, leaving a syndrome of dysarthria, dysphagia, involuntary laughing and crying, and bilateral rigidity. The disease usually occurs in conjunction with other evidences of arteriosclerosis and ordinarily in the aged.

The day following this examination this patient became quite stuporous, developed a marked retraction of the head, and rigidity in all extremities. Two tonic convulsions, each lasting for a few moments, occurred and the patient died of respiratory failure.

Necropsy revealed a mural thrombus of the basilar artery, beginning shortly after the junction of the two vertebrals, and completely occluding the left posterior inferior cerebellar artery, but with sufficient canalization to permit some blood-supply to the small branches of the basilar which were given off at the level of the mural thrombus.

REPEATED SPASM OF CEREBRAL VESSELS

Temporary interruption of vascular supply frequently produces a syndrome which indicates either a thrombus or an aneurysm of one of the arteries at the base of the brain. The following case illustrates the difficulty of differential diagnosis between these conditions:

A woman aged forty, who was seen for the first time on July 12, 1923, gave a history of having had an attack on December 15, 1921, which began with difficulty in swallowing, itching of the abdomen and face, and a slight swelling at the outer canthus of the left eye. Ten days after this itching began the right side of the face felt cold and one week afterward was paralyzed. The right arm was very tremulous, so that she could not write or feed herself, and considerable weakness was present in her right leg. Objective vertigo appeared and it was necessary for her to be kept in bed for seven weeks. She gradually recovered, but two weeks prior to her examination was suddenly seized with a vertiginous attack, with marked ataxia, numbness of the left side of the face, slight headache, and at the height of vertigo some vomiting.

Upon examination she showed an analgesia of the left side of the face, marked cerebellar ataxia of the left side, with inability to walk across the room, intention tremor of the left arm, adiadokokinesia, and dysmetria. There was diminution of sensation to painful and thermal stimuli on the right side. The Wassermann reaction on both blood and spinal fluid was negative, and no abnormalities were found in the spinal fluid.

A diagnosis of thrombosis of the left posterior inferior cerebellar artery was made. The symptoms and signs began to disappear after a period of three weeks, and she made a complete recovery within two months following the onset of the last attack.

In October 1926 I was called to see this patient again. She then gave a history of having had two attacks, one in 1924 and another in 1925. The first was characterized by the sudden onset of a peculiar feeling in the right side of the face with difficulty in speaking, which consisted of a paraphasia and right homonymous hemianopsia. This condition persisted for several months and then completely disappeared. The next attack consisted of the sudden onset of a right-sided hemiplegia, with complete aphasia, which condition likewise disappeared after several months. The last attack had occurred a week prior to my visit, and was characterized by a sudden onset of a right-

sided hemiplegia, paraphasia, and right homonymous hemianopsia.

Ophthalmoscopic examination was negative and there was no evidence of increased intracranial pressure. The abdominal reflexes on the left were brisk and no temporal pallor was seen in the disks.

The symptoms and signs indicative of widely scattered, diffuse, and discrete lesions immediately suggested the possibility of a multiple sclerosis, but the integrity of the abdominal reflexes and absence of central scotoma and temporal pallor, as well as the completeness of recovery from each attack, served to rule this condition out.

At the time of my first examination, when the diagnosis of thrombosis of the posterior inferior cerebellar artery was made, a note was made as to the possibility of aneurysm of the basilar artery. Both diagnoses were obviously wrong, as the pathology of either of these vessels could not produce the homonymous hemianopsia or the aphasia.

We were, therefore, confronted with a condition which at one time affected one and at another time another cerebral vessel. It was only at this time that the patient remembered that she had for many years suffered from a peculiar condition in her hands. Namely, that when exposed to cold or inclement weather, or when washing clothes in cold water, both hands would begin to tingle, burn, and shortly become blanched and painful.

She recovered from the last attack after a period of about six weeks, and reported to my office one morning with an attack typical of Raynaud's disease. The arterial pulses were almost imperceptible, the fingers were completely blanched, she experienced great difficulty in moving them, and suffered from numbness, tingling, and pain.

Although the diagnosis is unconfirmed by examination of her tissues, I am inclined to believe that we are dealing here with paroxysmal vascular spasms of the cerebral vessels, dependent upon a pathologic condition which is responsible for similar spasms in the peripheral vessels. A similar picture

frequently is presented by patients suffering from angioneurotic edema, and a number of cases have been reported of temporary hemiplegias associated with Raynaud's disease. In the absence of history of former attacks there would be no criteria for differentiating the condition from thrombus or aneurysm of the cerebral vessels.

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CUTANEOUS TUBERCULOSIS (SCROFULODERMA) AND TUBERCULIDS (ERYTHEMA INDURATUM, FOLLICLIS, ACNE SCROFULOSORUM) OCCURRING IN A CHILD WITH TUBERCULOSIS OF THE KNEE-JOINT

THE patient is a Greek girl, aged seven, who was admitted to the hospital on December 28, 1926, and the following history had to be obtained through an interpreter: There was nothing of interest in her history until April, 1926, when she developed a reddish, indurated nodule just below the outer malleolus of the left ankle. This lesion persisted unchanged and without tenderness for six months, when it broke down and formed the ulcer now visible. In September she developed a large, purplish, infiltrated, oval-shaped plaque on the right calf. This had shown some tendency to ulceration, but the latter subsided spontaneously before her admission. About the same time that the plaque appeared she had developed a disseminated eruption over the legs, thighs, and lower portion of the abdomen, and this eruption showed two types of lesions. On the legs only were scattered six to eight firm, pea-sized, reddish nodules, which had broken down in the center, while the more profuse element, involving the legs as well as the higher parts mentioned, consisted of pin-head-sized scaling papules. In October she had developed a marked swelling of the left knee, and two days before she entered the hospital there had appeared two pin-head-sized openings in the skin over the inner edge of the patella, from which there had been a thin, yellowish, rather scant discharge.

Physical examination reveals a fairly well-developed, moderately well-nourished, but anemic appearing child who is not

acutely ill and who is apparently not suffering any pain. She lies with the left knee in an attitude of slight flexion. The lungs and heart are normal, and aside from the features mentioned above there is nothing of note except a generalized adenopathy of mild degree, hypertrophied tonsils and adenoids, and a number of carious teeth, the permanent incisors showing a defective development of enamel.

With regard to the features which interest us, we found upon admission that the left knee was greatly swollen, somewhat tender when moved, and without any spasticity of the muscles. The overlying skin was of normal color and tempera-

Fig. 1.—Cutaneous tuberculosis, showing plaque of erythema induratum and folliclis.

ture, but presented two pin-head-sized openings over the inner aspect of the knee. There was a discharge from these, and this could be expressed readily by pressure. During the two weeks which have elapsed since her entry to the hospital these openings have enlarged until they are now apparent as two ragged, irregular ulcers with undermined edges, and with a bluish discoloration of the skin about them. They are separated from one another only by a narrow band of epithelium and doubtless will soon coalesce to form a single ulcer. They do not permit the passage of a probe to a depth greater than $\frac{1}{2}$ inch and no sinuses leading to the deeper structures are demonstrable.

The lesion near the left ankle is of similar type, but consists

of a single, kidney-bean-sized irregular ulcer with a ragged undermined edge, and with a zone of bluish discoloration about it. There is moderate infiltration of this zone, but no suggestion of involvement of the deeper tissues.

The large, bluish plaque on the calf of the right leg appears as an oval, hen's egg sized, rather deeply infiltrated lesion which has obviously been superficially ulcerative, but is now resolving, and presents adherent scale crusts at the sites of the former ulcers. Scattered over this leg are a few split-pea-sized, firm, reddish, fairly superficial nodules which appear to involve the epidermis and corium. They are in various stages of evolution, the fresh lesions presenting a small necrotic plug at the center, while the oldest lesions show a tendency to central atrophy with the formation of a smooth, depigmented scar surrounded by a zone of hyperpigmentation. Over the lower extremities and the lower portion of the abdomen is a fairly profuse eruption consisting of disseminated follicular, pin-head sized, accuminate reddish papules which are discrete and show no tendency to grouping. Some of these are crusted, while the older lesions have left pigmentation.

x-Ray examination of the chest shows no evidence of tuberculosis, and skiagrams have failed to show any evidence of bone or joint pathology of the left knee, leg, or ankle. The Pirquet test gave a strongly positive reaction. The Wassermann and Kahn tests are negative. The hemoglobin was 60 per cent., and the white cell count 9250 per cmm., with 26 per cent. lymphocytes, 23 per cent. large mononuclear, and 51 per cent. polymorphonuclear cells. Tissue taken from the edge of the ulcers on the knee was inoculated into a guinea-pig, but insufficient time has elapsed for a report upon this result. Microscopic examination of tissue from the same site, however, revealed a characteristic picture of tuberculosis. During the time that the patient has been under observation her temperature has been irregular, but rather consistently higher in the afternoon, and on three occasions has reached a height of 100° F. in the late afternoon.

The child was sent into the hospital from the orthopedic

dispensary with a diagnosis of tuberculosis of the knee-joint, and we were asked to see her in regard to the relationship of the cutaneous manifestations.

From the dermatologic standpoint the case is exceedingly interesting, presenting as it does four clinical types of lesions, all of which are recognized as of tuberculous origin. You will recall that the tuberculous manifestations of the skin are divided into two groups. The first of these comprises the types of true tuberculosis of the skin and includes lupus vulgaris, tuberculosis verrucosa cutis, and scrofuloderma. The latter variety is seen rather commonly as a result of invasion of the skin by tubercle bacilli from tuberculosis of underlying bones, joints, glands, etc., although it is occasionally seen independently of such underlying pathology. In this variety, described by the term *tuberculosis colliquativa*, there appear subcutaneous lesions of gummous type of tuberculous origin with later involvement of the overlying skin. In the present case the ulcers of the knee region are typically of the scrofulodermatous variety, but the lesions near the ankle, in which there is no demonstrable evidence of involvement of the underlying bones or joints, may be of the colliquative type.

The second group of cutaneous manifestations associated with tuberculosis consists of a number of clinical conditions which are known as tuberculids. This term was originally suggested by Darier to designate a number of conditions which were seen usually in tuberculous individuals, but in which the lesions did not present a microscopic picture of tuberculosis, and in which the tubercle bacillus was not demonstrable. The lesions are usually symmetric, show a tendency to recurrence in crops, run a benign course, and present a varied histologic picture, often suggesting tuberculosis. Originally it was thought that these lesions resulted from the action of toxins elaborated by the tubercle bacilli in active or latent visceral foci, and carried to the skin by the blood-stream. Later, theories were advanced that they were due to invasion of the skin by bacilli of lowered virulence, or by dead tubercle bacilli, inasmuch as it is occasionally possible to demonstrate the bacilli in the lesions.

During the past decade, however, it had been widely accepted that the tuberculids represent an allergic reaction on the part of the skin of a tuberculous individual. It is supposed that the skin of the patient, as the result of the pre-existing visceral infection, has acquired a state of hypersensitiveness toward the tubercle bacilli, and that when the organisms are carried to the skin from such a focus by the blood-stream, there to form microbic emboli, the sensitized skin reacts promptly, and in its attempt to destroy the organisms produces the non-specific granulomatous lesions which we recognize clinically as tuberculids. The rapid destruction of the bacilli which follows this reaction accounts for the infrequency with which the bacilli are demonstrated in the lesions. Thus, the tuberculids may be regarded, as suggested by Rist and Rolland, as spontaneous examples of Koch's phenomenon.

Although the number of conditions which are included among the tuberculids is large, many of them cannot be proved as deserving of classification in the group, while it is conceded that others are not always dependent upon tuberculosis for their genesis, but may result from other causes as well. The most important members of this class are the papulonecrotic tuberculids (acnitis and folliclis), erythema induratum (Bazin's disease), and lichen scrofulosorum. Among the less well-established members are the sarcoids, lichen nitidis, lupus erythematosus, pityriasis rubra, etc.

In this case we have a good demonstration of three of these conditions associated with tuberculosis, as the large purplish plaque is typical of erythema induratum, the smaller lesions with the necrotic centers are characteristic folliclis, while the more wide-spread eruption of follicular papules corresponds in every detail with the description of one of the less common tuberculids, acne scrofulosorum.

This case is of particular interest in that it well illustrates an often neglected diagnostic index. In this instance the tuberculids were present for some months before the knee-joint became involved, and had the patient been seen when the early cutaneous manifestations alone were present, and a proper

antituberculous régime instituted, the joint involvement might have been avoided. Not infrequently the recognition of tuberculids in a patient will lead to more careful examination for tuberculosis, and may point to the proper diagnosis of an obscure illness dependent upon such infection.

The patient is receiving, in addition to the establishment of a proper régime and orthopedic care of the knee, generalized ultraviolet light baths. Treatment with a carbon arc lamp would be preferable, but is not available. She is already showing rapid improvement as a result of this treatment. Other agents which are useful in the treatment of the tuberculids are arsenic, particularly in the form of arsphenamin, *x*-ray therapy and, in selected cases, tuberculin.

XANTHOMA TUBEROSUM MULTIPLEX OCCURRING IN A WOMAN WITH LATENT SYPHILIS

THIS patient, who is an American woman, aged thirty, first came under observation in January, 1926, when she entered the clinic because of an eruption involving the extensor surface of the elbow, the wrists, palms, and patellar regions. She stated that these lesions had begun to appear some seven months earlier, when she noticed several pin-head to slightly larger yellowish nodules over each elbow. These gave rise to no symptoms, and were discovered accidentally. They gradually increased in size and new lesions developed. Some three months after the first lesions were seen about the elbows she noticed some similar nodules on the palms and backs of the fingers over the knuckles, and also remarked that the lines of the palms and of the flexor surfaces of the wrists were showing a peculiar yellowish discoloration.

Before she came to the clinic her physician had suggested that she have a Wassermann test made, and a strongly positive reaction was reported. This was confirmed in our laboratory, although she had received three courses of mercurial injections and one course of eight injections of arsphenamin. She has been married for ten years and was unable to offer any history which might give a clue as to the date of the syphilitic infection. Her past history otherwise was without interest except for the occurrence of one stillbirth, her only pregnancy.

Physical examination revealed nothing of note aside from the skin lesions. There was no adenopathy and nothing else suggestive of a specific infection. The hemoglobin was 85 per cent., red blood-cells 5,700,000, white blood-cells 14,250, with 36 per cent. lymphocytes, 4 per cent. large mononuclears, and 56 per cent. polymorphonuclear neutrophils. The urine was negative and phenolsulphonephthalein excretion normal. The bromsulphthalein test of liver function showed 20 per cent. of the

dye remaining in the serum after thirty minutes, when normally there is no dye remaining at the end of this time. The non-protein nitrogen content of the blood was 55 mg. per 100 c.c. of blood, and the blood-sugar 100 mg. per 100 c.c. of blood, while the cholesterol content was 250 mg. per 100 c.c. of blood.

The cutaneous lesions involve principally the extensor surfaces of the arms symmetrically, extending from about 2 inches

Fig. 2.—Xanthoma tuberosum multiplex, papular and nodular lesions.

above the elbow to a point half-way down the forearm. Here they appear as chamois yellow to reddish-yellow papules and nodules varying in size from that of a pin-head to that of an almond, the larger lesions obviously resulting from coalescence of smaller papules. They are moderately firm, and while the smaller lesions are round or oval, and but little elevated, the larger masses are less regular in outline and considerably elevated. There are no subjective symptoms unless the lesions

are struck, when they exhibit some tenderness. Similar lesions, all of the small papular type, are present on the palms, over some of the knuckles, and over the patellar region. In addition, the linear markings of the palms and wrists show strikingly because they are the seat of a linear deposit of a saffron yellow infiltrate.

This picture is perfectly characteristic of the condition known as xanthoma tuberosum multiplex, a disorder which is at times

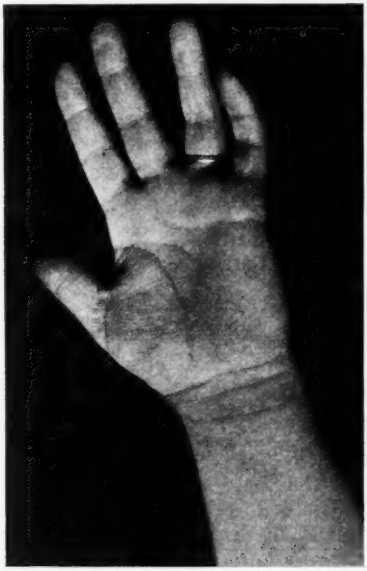

Fig. 3.—Xanthoma tuberosum multiplex, showing striæ of palms and wrist.

more or less generalized, but which has as its sites of predilection the extensor surfaces of the elbows and knees, and the hips, while striated lesions are commonly seen on the palms. The characteristic lesions are papules and nodules, but in some cases large tumors may be formed. In rare instances the mucous membranes of the mouth, larynx, esophagus, bronchi, and conjunctiva are involved. In some instances other parts, as the tendons and cornea, have been affected, and Weidman and

Walker have recently described a case with involvement of the lungs, liver, skull, pineal body, pituitary, and tuber cinereum.

It is generally accepted that the lesions represent, as Wile and Pollitzer pointed out, an irritative connective-tissue hyperplasia, in which the extravasation of cholesterol-fatty-acid present in the blood serves as a stimulus. The disorder is seen most commonly in association with diabetes mellitus and liver disease, although rheumatism, syphilis, and other diseases have been suggested as having a possible relationship. Within recent years the major interest in the study of xanthomatosis has been devoted to the study of the cholesterol content of the blood. Harrison and Whitfield have recently studied from this standpoint the cases reported in the literature, including the diabetic and xanthelasma types as well as the tuberose variety, and found an increased cholesterol content, usually of high degree, in 30 out of 40 cases, an average of 75 per cent. Of the 10 cases in which there was no hypercholesterolemia 6 were of the xanthelasma type. They suggest that pancreatic dysfunction may be a responsible factor in some cases.

Our patient shows, as I have said, a definite hypercholesterolemia, and we have been interested in a possible relationship between this finding and the existence of syphilis, although Rosen and Krasnow, in a recent study, found no evidence of a tendency toward hypercholesterolemia in syphilis. We have given her, during the past year, three courses of antisyphilitic therapy, employing neo-arsphenamin and bismuth, but have been unable to effect any change in either the Wassermann reaction or the cholesterol content of the blood, and the xanthoma lesions, as was expected, not only have not retrogressed, but new lesions have appeared, and the older ones have grown larger. Little is to be expected from the treatment of this condition unless one resorts to surgery or the use of destructive measures, such as trichloracetic acid or the use of a 25 per cent. salicylic acid plaster. Recently it has been suggested that injections of iodipin are of benefit in this disorder, and we have employed this drug for the past two weeks in this case. It is, of course, too early to expect any results as yet.

RECURRENT SECONDARY SYPHILIS OF THE SKIN AND MUCOUS MEMBRANES

THIS young man is twenty-six years old. In 1922 he developed a generalized eruption which was apparently typical secondary syphilis, and he states that at that time his Wassermann reaction was reported as strongly positive. He is unable to recall having had any primary lesion. He received intensive antisyphilitic treatment, as during the succeeding two years he was given seventy injections of neo-arsphenamin and about 100 intramuscular injections of some mercurial salt, probably of soluble type. The eruption cleared up readily at the beginning of treatment, but although he is not aware of the results of the Wassermann tests made during this period, it is probable, because of the intensity of the treatment, that the Wassermann reaction remained positive. He states, however, that the reaction was negative when the treatments were stopped. During the succeeding year he felt well and had no trouble of any kind, but he then complained of becoming tired easily, of requiring more sleep than usual, of constipation, and "stomach trouble." He again consulted his physician, and as the Wassermann reaction on both the blood and spinal fluid was negative and there was no evidence of active syphilis, he was treated only for his other complaints. Three months later, however, he developed another eruption, this time less generalized than was the first, together with some lesions of the oral mucosæ, and his serologic reaction was again positive. He then received twelve injections of neo-arsphenamin and the same number of injections of mercury salicylate. The eruption and the mucous membrane lesions persisted in spite of this, and he consulted another physician. The Wassermann reaction was still positive, and during the next eighteen months he was given eighteen injections of neo-arsphenamin and forty injections of mercury. During this period the recurrent eruption improved, but never

cleared up completely. During the past year he has received no treatment, and six months ago the Wassermann reaction was negative. During the past four months the eruption on the body has become more extensive, the mucous membrane lesions have been profuse, and five weeks ago the serologic test gave a positive result again. He consulted his physician, and was advised to take further treatment with neo-arsphenamin and mercury, but as he had become discouraged he came to the clinic two weeks ago for advice.

At that time he presented a profuse eruption, the lesions being indurated, dull brownish-red papules arranged in annular or archiform configurations, and showing no tendency to ulcerate or produce scars. There were probably 100 of these groups scattered over the trunk and extremities. They constituted a characteristic recurrent syphilitic eruption of the early type, as in these recurrences there is a decided tendency for the lesions to arrange themselves in an annular configuration. In addition, the tonsils, fauces, and soft palate presented a number of angry appearing mucous patches.

It was decided to treat the patient by means of bismuth injections, and he was given one injection of potassium bismuth tartrate, 0.2 gm., two weeks ago and another one week later. Upon examination today we find that the lesions, which persistently resisted treatment with neo-arsphenamin and mercury have responded readily to the bismuth medication. The mucous membrane lesions have disappeared entirely, while the cutaneous eruption is evidenced only by the presence of some residual pigmentation.

This patient is not presented because of the favorable effect of the bismuth, but because the case furnishes a valuable object lesson in regard to the present-day situation in the therapy of syphilis. Since the early days of arsphenamin therapy we have been confronted with cases of syphilis which present early relapse, usually involving the skin, mucous membranes or central nervous system, and we have also become familiar with those cases in which the Wassermann reaction is persistently positive or tends to relapse. Unfortunately, there is a decided

increase in the proportion of cases of this type, as is agreed by practically all observers. Consequently, too much emphasis cannot be placed upon the necessity for prolonged and intensive treatment in early syphilis, with practically continuous treatment with one drug or another during the first year. One cannot complain that in the present case this was omitted, but the subsequent history of the treatment does indicate a disregard for one principle of therapy. It should be known by everyone who attempts to treat syphilis that, with the greater variety of therapeutic agents now available, it is possible when we fail with one drug or combination of drugs, to change our method of attack. It is recommended that the physician, when confronted with a case of resistant or relapsing syphilis, take occasion to consider the situation with a view to modifying or recasting completely his therapeutic procedure. Not infrequently, when neo-arsphenamin has been employed unsuccessfully, a change to arsphenamin or sulpharsphenamin will give results. If the insoluble mercurials have been employed, a soluble salt may prove more useful. It is in this type of relapsing and resistant syphilis particularly that bismuth therapy finds a place, as it enables us to attack the disease from a fresh therapeutic angle. This case demonstrates the efficacy of such a change. Cases with a therapeutic history such as this show the necessity for urging the physician to take advantage of the opportunity to revise the treatment of any case when the course of the disease makes it advisable.

BULLOUS IODODERMA. RAPID RECOVERY FOLLOWING THE USE OF INTRAVENOUS INJECTIONS OF SODIUM CHLORID

THIS negress is thirty years old and came to the clinic eleven days ago. She presented an eruption of pustular and impetiginous type over the face, with a small, vegetating plaque on the nose, a pustular eruption of acneform type over the chest and back, and on the forearms a severe bullous outbreak of about two weeks' duration. The acute inflammatory appearance of the acneform elements, as contrasted with the more sluggish appearance of the lesions of acne vulgaris, and the vegetative character of the lesion on the nose, at once called attention to the probability of this condition being dermatitis medicamentosa, probably from iodids or bromids. On questioning the patient we elicited the information that she had been taking for several weeks a proprietary medicine which contains potassium iodid.

Eruptive manifestations following the taking of iodids or bromids are not at all uncommon, and because of the variety of forms in which the idiosyncrasy manifests itself, diagnostic pitfalls are often encountered. Although the common type of eruption is a papulopustular or pustular acne, we may encounter erythematous, vesicular, urticarial, furuncular, carbuncular, erythema multiforme or erythema-nodosum-like manifestations, and occasionally, particularly from iodids, a bullous variety. Perhaps the most confusing cases are those in which the lesions assume a vegetating character, when they are described by such terms as mycotic, granulomatous, fungating, or frambesial. In such instances the disorder may be confused with mycosis fungoides, syphilis, tuberculosis verrucosa cutis, blastomycosis, sarcoma, or other diseases.

The amount of the drug necessary to give rise to eruptive manifestations varies considerably, as occasionally only a few

small doses have been taken before the outbreak occurs. If the nature of the eruption is not discovered and the use of the drug is continued, it may lead to serious and permanent disfigurement and grave results, particularly in the cases of children. It is worthy of remembrance that with the iodids and bromids the eruption may persist for a long time after the withdrawal of the drug, while in some instances the eruption does not begin until the use of the medicine has been discontinued.

Fig. 4.—Iododerma, showing papulopustular lesions of face and vegetating patch on nose.

It is agreed by many observers that the use of small doses of arsenic is of value in avoiding the undesirable effect of these drugs, and more recently Hubschmann, who has found the calcium content of the blood diminished in such cases, has suggested calcium therapy. From the therapeutic standpoint, however, the most interesting suggestion has been made by Wile and his co-workers. In an experimental study they showed that the ingested bromid readily displaced the chlorid in the blood.

They felt that it seemed quite likely that stored-up bromids could be flushed out of the body by mass action of relatively large amounts of chlorid. They found that patients with bromism when treated with intravenous injections of sodium chlorid, showed an apparent liberation of bromid in the urine, together with rapid improvement of the mental and cutaneous symptoms. They advocated the injection of 100 to 400 c.c. of decinormal

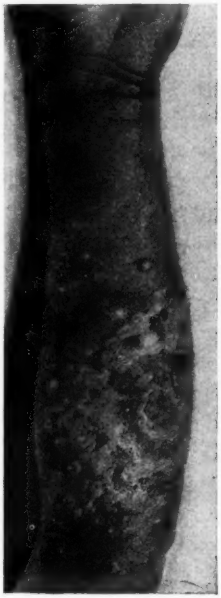

Fig. 5.—Iododerma, bullous dermatitis of forearm.

Fig. 6.—Iododerma, vegetating patch on hand.

salt solution at intervals of two to three days, and a number of other workers have observed excellent results from this method of treatment.

Our patient was given an injection of 150 c.c. of saline solution nine days ago, and 200 c.c. six days ago. There was great improvement when she returned for her second injection, and by the seventh day the eruption had practically disap-

peared. We were unable to study the case from the standpoint of determining the iodid content of the urine before and after treatment, but the clinical course of the disorder has been very short as contrasted with the average case of ioderma. This method of treatment is perhaps not necessary for the milder cases, but it is worthy of use in the more severe types, particularly in the cases of bromism with mental symptoms.

CLINIC OF DR. SAMUEL J. TAUB

COOK COUNTY HOSPITAL

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS COMPLICATED WITH DIABETES MELLITUS

ARTIFICIAL pneumothorax is the greatest advance in the therapy of tuberculosis which has been employed within the past two decades and its importance is all the greater in the cases where it is applicable. Before I discuss the use of this treatment in pulmonary tuberculosis complicated with diabetes a review of the indications and contraindications in a general way is advisable, as the success of pneumothorax treatment depends very largely upon the proper selection of cases. The typical case is one which has extensive disease in one lung, with the other lung free or slightly diseased. Just how much disease is necessary to justify collapse cannot be definitely stated; but, as a general rule, it may be said that unilateral disease, whether slight or extensive, which is progressive or shows no tendency to heal is suitable. The condition of the good lung is of greatest importance in deciding for or against pneumothorax. The non-collapsed lung has to carry on the whole respiratory function.

It is often true that it is doing so largely before pneumothorax is begun, as the diseased lung, by the very presence of the disease, is incapable of aiding much in respiration, and in these cases activity in the worse lung is adding to the strain on the better one, which is, in reality, given less work to do by rendering the disease quiescent. Even slight disease in the opposite lung becomes arrested and signs disappear after collapse of the more diseased lung.

Disease at the base of the better lung and definitely acute disease in any part are considered as contraindications to pneumothorax.

Dyspnea is usually a contraindication, but in some cases this is toxic and is relieved when the source of the contraindication is rendered inactive.

Cyanosis is always a contraindication. Among the acute forms of tuberculosis miliary disease is not amenable to this treatment. The pneumonic form has not shown any benefits from this procedure. The acute bronchopneumonic form involving one lung often shows marked improvement.

The most favorable type is that in which the lung is filled with coarse moist râles with or without cavity.

Large and uncontrollable hemoptypses, when definitely coming from one lung, are often controlled by compression. In such cases it is advisable to use large amounts of air, up to 1000 c.c., at the initial operation in order to stop the bleeding.

Asthma and emphysema contraindicate pneumothorax therapy. Well-marked cardiac lesions are contraindicated, as compression of one lung puts a strain on the right side of the heart.

Advanced nephritis is a contraindication unless it is toxic in origin, in which cases the renal condition may be improved.

Diabetes is always a grave complication, yet remarkable results may be obtained in the proper selection of cases and especially if pneumothorax is instituted early.

The Apparatus.—Each apparatus consists of two graduated bottles connected by tubing, one of which contains air and the other fluid. When the fluid flows into the empty bottle it displaces the air which is pressed into the pleural cavity through a rubber tube and the needle. The manometer indicates the intrapleural pressure. As soon as the needle enters the free pleural cavity, a negative pressure will be registered due to the vacuum in the pleural space. With inspiration and expiration the manometer will register the changes of the air pressure in the pleural space. With the aid of the manometer one can tell whether one is in the pleural cavity and whether the adhesions are extensive or slight. If in going through the pleura the manometer does not register, one of several things may have occurred. Either the needle is not entirely through the chest wall, or it may have gone on through the visceral pleura into the

lung. Adhesions or the presence of fluid in the chest will also interfere with the free fluctuation of the manometer.

The use of nitrogen gas has been discarded and atmospheric air is now used, as it was found that there is a rapid diffusion of the two gases—nitrogen and oxygen.

The Operation.—Pneumothorax should be considered as a surgical procedure and strict asepsis is necessary. The site selected for injection is cleansed with iodin and alcohol. Sterile towels, thorough scrubbing of the operator's hands, and the use of rubber gloves must be insisted on.

A novocain solution of ½ per cent. is used as a local anesthetic. The axillary line in the sixth or seventh interspace is selected because the chances of entering are better. Small amounts of the gas, 200 to 400 c.c., and frequently repeated, should be given. The treatment must always be finished with a negative pressure. Pleural adhesions are the cause of many failures to attain a good compression of the diseased lung. A collapse must be complete in order to give the most beneficial results, although many patients will do well with only a partial collapse.

If the pleural space is found to be free and air is allowed to flow, the manometer should be observed at each 50 c.c. of air given. A small initial dose of 200 to 350 c.c. of air should be given.

After the initial pneumothorax instillation the next few treatments should be given every third or fourth day, going into the same space as before. The amounts of gas given at refills should not be large, depending, of course, upon the intrapleural pressure. After complete collapse of the lung, as guided by manometer, fluoroscopic and physical examination, the frequency of refillings should be diminished to about once a month.

Symptoms.—Immediately upon completion of the operation the patient feels a slight difficulty in breathing. When the gas separates adhesions slight pain is experienced. The temperature usually drops to normal if a good compression is obtained and remains there unless complications set in. Rapid improvement in the cough is noted, especially when a cavity has been successfully compressed. Even a partial compression may result in

a disappearance of toxic symptoms, such as night-sweats, cough, and high temperature.

Complications.—Pleural shock and air embolism may occur during the operation, but these complications are comparatively rare. Pleural effusion is a very common and rather serious complication which occurs in from 10 to 50 per cent. of the cases. If the fluid is small in amount it is generally transitory in character, lasting only, a few days, without producing any outward symptoms. A sudden elevation in temperature in a patient doing well under pneumothorax treatment often means the appearance of fluid. The fluid will be found to be clear on aspiration, but will become cloudy or purulent if a mixed infection sets in. The treatment of this condition depends upon the symptoms found in each case. If there is absence of fever or dyspnea no intervention is necessary. When there is a rapid accumulation of fluid causing cardiac or respiratory embarrassment the indications call for aspiration and replacing with air.

I will now discuss the use of this treatment in 2 cases of pulmonary tuberculosis complicated with diabetes mellitus.

Case I.—Mrs. C., age thirty-seven years, widowed, entered the Cook County Hospital on April 18, 1926 with the following complaint: Loss of weight of 82 pounds in one year, cough of four weeks' duration.

Onset and Course.—Patient states that she was in good health until one year ago, when she began to lose weight slowly, but steadily. One year ago she weighed 200 pounds and today 118 pounds. A cough has been present for one month, usually worse in the morning. Never coughed up blood. Excessive thirst has been present several months. Pain in the right chest, especially after cough has been present about two months. No dyspnea is present. Night-sweats have been present about two months. Edema of ankles occurs after standing on her feet a long time. No palpitation is present. Bowels are constipated, for which she takes pills every other night.

There is some frequency of urination during the day and

nocturia occasionally. Menstrual history is negative and there is nothing of importance in either her past or family history.

Physical Examination.—Reveals a rather light colored adult negress who does not appear acutely ill, but has a chronic cough. Pulse is 100, temperature 100.8° F., respirations 24, and blood-pressure 120 systolic, 80 diastolic. Head and scalp negative.

Physical examination is entirely without import except for slightly enlarged tonsils and the findings in the chest. Lungs show impaired resonance over the right upper lobe, harsh bronchovesicular breathing, and many fine and medium moist râles scattered throughout right upper lobe and upper one-third of right lower lobe; below the clavicle is a small area of intense bronchial breathing and whispered pectoriloquy and showers of moist râles. There were no abnormal findings in the left lung.

Heart apex-beat not visible, rate and rhythm regular, no murmurs. Right heart border is substernal, left border in midclavicular line, and the upper border is at the fourth rib.

Laboratory Findings.—*x*-Ray examination on April 12th revealed a caseous and infiltrative tuberculosis involving the right upper lobe and the upper part of the right lower lobe, with a few contained cavities about the level of the second intercostal space. The trachea was displaced to the right.

Urinalysis on April 9th showed the following: Catheterized specimen: amount, 75 c.c., straw color; specific gravity, 1044; reaction acid; no albumin; sugar present, ++, and acetone present, ++. On April 10th a twenty-four-hour specimen showed amount 2050 c.c., straw color and cloudy; specific gravity, 1040; acid reaction; sugar, 78.8 grams; acetone, +.

Blood chemistry on April 8th showed uric acid, 3; sugar, 0.315; acidosis, 52.

Wassermann test on the blood was negative. There were 6900 white blood-cells and 60 per cent. hemoglobin.

Sputum examination revealed the presence of tubercle bacilli.

A diagnosis of diabetes mellitus and pulmonary tuberculosis of the right upper lobe with evidence of cavitation and involvement of upper one-third of right lower lobe was made.

On April 10th patient was started on a diet of carbohydrate 20, protein 29, and fat 24. On the 18th she was sugar free and the diet was increased to carbohydrate 34, protein 53, and fat 94, giving a glucose value of 74. F. A/G ratio of 1.148. On the 22d she was sugar free and the diet was changed to carbohydrate 55, protein 53, and fat 136, or 1656 calories. She remained sugar free until a diet of carbohydrate 102, protein 70, and fat 239, or total calories of 2939, was reached, when sugar was excreted in the urine. The diet was reduced to 2094 calories, and insulin, 15 units twice a day, was given. The patient continued toxic, was excreting sugar daily, and becoming emaciated and weak.

On May 18th a pneumothorax was done in the right chest and 250 c.c. of air introduced, using the Robinson apparatus. The pneumothorax was repeated at intervals as follows:

	C.c. of air.
May 21st	300
May 26th	300
June 3d	350
June 10th	350
June 18th	350
June 26th	350

She was sugar free from May 21st and the diet was increased to 2633 calories, in the proportion of carbohydrate 92, protein 60, and fat 225. By July 10th a complete collapse was effected and the urine remained sugar free without the use of insulin. On July 15th the diet was increased to 2839 calories, consisting of protein 70, carbohydrate 102, and fat 239. On July 10th the urinalysis showed no albumin or sugar. Blood-sugar was 0.125. A steady gain in weight was noticed and there was no cough, night-sweats, or other toxic pulmonary symptoms.

Case II.—The second patient, Mr. A. D., aged forty-two, was admitted to the Cook County Hospital May 5, 1926 complaining of pain in the right chest, cough, weakness, and lassitude, and the loss of 50 pounds in weight. These complaints dated back five months.

The patient stated that he caught cold about five months previously and the cough continued. Sputum was blood tinged at times. For three months he had had night-sweats. He had headaches occasionally. Cough was productive of a yellowish and blood-tinged sputum. He had pain in the right chest, especially after coughing. Shortness of breath was present on exertion.

With the exception of a nocturia four or five times each night his history was negative. There was nothing in his past history except malaria twenty years previous, and gonorrhea and chancre eighteen years previous.

Physical examination revealed an obese white male, forty-two years of age, who was not acutely ill. Temperature was 99° F., pulse 108, and respirations 24.

Tonsils were enlarged and pharynx injected. There was a lagging of the right side of the chest; dulness in right upper lobe; bronchovesicular breathing and many moist râles in the upper lobe. There was a cavity in the right upper lobe below the clavicle. Heart borders were within normal limits, and the heart tones were faint but regular.

Physical examination was otherwise unimportant.

Laboratory Findings.—Urinalysis showed specific gravity 1038, sugar present ++, no albumin or acetone. Blood-sugar 0.280. Blood Wassermann was negative.

The patient was put on a diet of 1540 calories, consisting of protein 50, carbohydrate 20, and fat 140, with a F. A/G ratio of 2.3, and continued to excrete sugar.

On July 20th he had a pulmonary hemorrhage of about 500 c.c., and a pneumothorax was begun with 300 c.c. of air in the right pleural cavity. On July 25th 550 c.c. of air was introduced and there was no further bleeding. On July 29th he was sugar free for the first time and the general condition had improved considerably. The diet was increased to carbohydrate 40, protein 50, and fat 105. On July 30th 450 c.c. of air were given and on August 14th, 500 c.c. On August 24th another 500 c.c. was given. He had been sugar free during this entire period and the toxic symptoms were absent. On September

1st, 800 c.c. of air were given and the diet changed to carbohydrate 70, protein 60, and fat 170, a total of 1950 calories. On September 15th blood-sugar was 0.120. On September 26th 500 c.c. of air were given and the patient continued to gain in weight from 156 pounds on admission to 178 pounds, and was sugar free.

SUMMARY AND CONCLUSIONS

Two cases of far advanced pulmonary tuberculosis with cavitation and complicated with diabetes mellitus were treated with artificial pneumothorax with marked beneficial effects on both diseases and a definite raising of the sugar tolerance. The raising of the sugar tolerance is definitely due to the control of the toxic pulmonary symptoms by collapse of the diseased lung, and thereby holding in check the spreading of the tuberculosis.

CLINIC OF DR. SAMUEL M. FEINBERG

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ASTHMA DUE TO HOUSE DUST

TODAY I am going to present to you 2 or 3 cases of bronchial asthma which represent a subgroup of this condition, the presence of which has only been recognized in the last few years, but the importance of which is growing steadily. I am referring to asthma due to hypersensitiveness to dust.

Before these patients are presented it will be advisable to make a few introductory remarks on this subject. It has long been recognized that asthma is caused by hypersensitiveness of the individual to a specific protein. The presence of hypersensitiveness is determined by placing the suspected extracted protein on a scratch in the epidermis, or injecting a small amount of the dissolved protein intradermally. When hypersensitiveness exists it will manifest itself in the form of an urticarial wheal within five to thirty minutes.

By these means it has been repeatedly shown that there are a large number of substances capable of causing hypersensitization. These substances, or antigens as they are commonly termed, can be classified into several groups depending on their portal of entry into the body. These are the inhalants, ingestants, contacts, and intrinsics. The substances which cause hypersensitiveness by contact are relatively few and need not be discussed at this time. By the intrinsic group we mean bacteria, either in the bronchial mucosa or elsewhere, and also other metabolic substances of more doubtful incrimination. The ingestants comprise chiefly foods of various kinds, condiments, and beverages. The inhalants, which constitute the most important class, include the animal hairs, danders, feathers,

furs, pollens, and miscellaneous substances, such as orris root, sawdust, tobacco, and many others.

There is a great percentage of asthmatics who have been found hypersensitive to dust. Now, it may be said at the outset that by hypersensitiveness to dust we do not mean the mechanical irritation caused by the dust. It is a common observation that asthmatic individuals, no matter what the substance to which they are sensitive, will frequently have their condition aggravated by house dust, street dust, coal smoke, fog, etc. This may be entirely mechanical and non-specific. A normal individual may also show some degree of pharyngeal or bronchial irritation when exposed to these mechanical irritants. However, it has been definitely proved that asthmatics may be specifically sensitive to dust, particularly house dust. The proof of this assertion rests on the demonstration of several facts. First, some patients have asthmatic paroxysms when exposed to the dust of certain houses or a particular room, and can inhale other dusts with impunity. Second, a positive skin test is obtained in these individuals. Finally, these patients respond to treatment with the same dust extract.

It is not at all surprising that hypersensitiveness to the dust proteins is so common when we consider that house dust constitutes an aggregate of many objects in the home in a finely divided state. These objects which give off dust readily are feathers, furniture stuffing, furs, hairs used in clothing and upholstering of furniture, rugs, and animal pets. Each one of these substances individually has been shown to be a cause of hypersensitiveness. It is but natural to find, therefore, that the dust as collected by the house sweeper gives positive skin tests and can be shown clinically to be an etiologic factor in the asthmatic paroxysms.

On the other hand, there are many cases of bronchial asthma which show no positive skin reactions to any of the extracts representing all known objects in the patient's home, nor to any other extracts for that matter, and who still give a positive test to house dust extract. These instances are more difficult to explain. For our purpose it is sufficient to realize that such

instances do exist, that we are able to recognize them by their skin reaction, and that we are able to treat these patients successfully with the same dust extracts.

The percentage of therapeutic and diagnostic results is better if the patients are tested and treated with dust extracted from their own home. On the other hand, a stock extract prepared from the dust of several homes usually give good results.

The preparation of these extracts will be described in brief. The patient is instructed to collect the dust from his home by means of a vacuum cleaner. It is best to collect samples from representative parts in the home: from the living-room rug, furniture, bed springs, dining room, etc. The usual quantity of dust used by me for extraction is 10 gm. This is extracted several times with ether until all fats have been removed. To the residue is added 100 c.c. of an aqueous solution of sodium chlorid, 0.5 gm.; sodium bicarbonate, 0.252 gm.; phenol, 0.4 gm. This is allowed to stand for twenty-four hours at room temperature. The mixture is then filtered through filter-paper and the clear filtrate sterilized by Berkefeld filtration. Sterility of this extract is further assured by making cultures, both aërobic and anaërobic. When the extract has been found sterile it is ready for use.

Treatment is commenced by giving two doses weekly, beginning with 0.05 c.c., 0.1 c.c., and then increasing every dose by 0.1 c.c. until 1 c.c. is reached. Then 1 c.c. is given once a week until the patient has been free from asthma for a considerable length of time. Due to varying degrees of hypersensitiveness and individual factors departures from the outlined schedule are often necessary.

The cases here presented are representative of a large group of similar cases under my care, and illustrate the type of cases, the method of diagnosis, and average results of treatment.

Case I.—E. H., colored woman aged thirty-seven, housewife, was admitted to the clinic on October 1, 1925, with the following complaints and history:

Complaints on admission were dyspnea, wheezing, and cough

coming on in attacks about once monthly and lasting about a week. The attack on admission was of five weeks' duration. Onset of complaint was in 1922 while working as a hotel maid. There is no seasonal variation in the asthma and no cough between attacks. She has not had any change of environment since onset of attacks. The patient has never had any household pets, but has five different kinds of ferns.

Appetite is good; there are no digestive disturbances; she takes a cathartic once weekly. She has complained of frequent urination and headaches for the last five weeks.

Previous illness constitute pneumonia at six months of age; polyarthritis from thirteen to fifteen years of age; measles at age of nineteen; frequent colds; otitis media six weeks before admission; no operations; venereal history negative. She has 2 children living and well, and has had one miscarriage at six months.

Family History.—Father, mother, 3 sisters, 3 brothers living and well. No history of asthma, hay-fever, urticaria, or eczema in family. Habits are negative except for a little wine occasionally and a cathartic once weekly.

Examination on admission showed a well-nourished colored woman, weighing 200 pounds. The pulse-rate was 96; temperature, 99.6° F.; blood-pressure, systolic 120, diastolic 89. The tonsils showed infection, as also did several teeth. The thyroid was moderately enlarged and soft. Musical wheezing râles were heard over the entire chest. No other positive lung findings were present. The cardiac apex impulse was not visible, the borders were normal, the tones clear; there were no murmurs. Abdominal examination was negative and the reflexes were normal.

The urine showed no abnormal findings. The blood examination made a month after the first admission showed 2 per cent. eosinophils. The blood Wassermann test was negative.

Skin tests were begun on October 6, 1925, using the cutaneous method. Slight positive reactions, from + to ++, were obtained to duck feathers, crab meat, lobster, salmon, shrimp,

and orris root. Patient then disappeared until March 23, 1926. At that time she complained of a continuous asthmatic state lasting two weeks. On March 25th intracutaneous test was made with my stock house-dust extract No. 1, using 0.01 c.c. A strongly positive reaction (++++) resulted.

Treatment was begun by subcutaneous injection of the dust extracted twice weekly beginning with 0.05 c.c., then 0.1 c.c., and then increasing by 0.1 c.c. until 1 c.c. was reached. Then 1 c.c. injections were given weekly. There was steady improvement, and two weeks from the commencement of the treatment the patient was free from asthma. However, she discontinued treatment for a month, during which time she had a slight recurrence. Further treatment again made her asthma free. Today (January 24, 1927) patient is free from asthma and has been thus for several months. It is noticed, however, that the intracutaneous test which I have made is still positive, a rather common finding even after clinical results have been successful.

Case II.—T. O., a white girl, age fourteen, was admitted to the clinic on October 8, 1925, with the following history:

Complaints on admission are cough, wheezing, dyspnea, and sneezing coming on in attacks about once in three weeks and very often at much shorter intervals. Attacks usually last one to three days. First paroxysm occurred in autumn of 1921; now attacks occur throughout the year. Paroxysms are more apt to occur at night. Partial relief is obtained by inhaling medicated vapors. She has never been free from symptoms for any long period. The only household pet is a dog.

Previous illnesses constitute pneumonia three times, the last attack occurring two years ago, whooping-cough, measles, and mumps.

The family history is negative for asthma, hay-fever, urticaria, or eczema.

Physical examination shows a slightly undernourished girl, weighing 97 pounds. The pulse-rate is 100; temperature 99.4° F.; respirations 22. Her tonsils are infected. The teeth are in good condition. There are no abnormal lung findings. The heart

borders are normal; the tones are clear. The abdomen is negative.

The urine is normal and the blood-count shows a normal picture with 1 per cent. eosinophils. The Wassermann test is negative.

Skin tests were begun on November 7th, and all proteins, totalling about 200, gave negative results, with the following exceptions: camel hair + to +++; house dust ++++.

Treatment was begun with house-dust extract on January 5, 1926. At that time the patient had been having constant dyspnea for several weeks. Improvement began after the second injection and continued. Treatment was carried out until May 11th, the patient receiving a total of twenty injections. During this time she had only two very slight wheezing spells of short duration occurring early in the course of treatment. The patient discontinued treatment on May 11th without our consent, and between that date and today (January 24, 1927) she says that she has had two moderate attacks of asthma.

Case III.—J. W., white boy, eight years of age, was admitted to the clinic on May 28, 1925, with the following history:

The complaints are sneezing, watery discharge from nose, wheezing, and shortness of breath coming on in attacks every few days. The first attack occurred in August, 1924. These symptoms occur at any time of the year and begin chiefly at night. There is no cough between attacks. He plays with cats and dogs frequently.

The previous illnesses are pneumonia when seventeen months old, followed by otitis media. The family history is negative for allergic disease.

Examination shows the boy to be about 10 per cent. underweight. The throat is normal. Wheezing râles are found in the chest, but no other abnormal lung findings. The heart and abdomen are negative.

The urine examination and blood-count show normal findings. *x*-Ray of the chest shows slight increase in hilus shadows, but no other changes.

Skin tests at that time were all negative except for for two

or three 1+ reactions which were not considered significant. No tests were made with dust extract at this time, as we had not yet begun to use it. The boy was then returned to the pediatric department.

On June 29, 1926 the patient was again referred to the Asthma Clinic. At that time he was wheezing and had shown no improvement. Intracutaneous test at this time with house-dust extract gave a ++++ reaction. Treatment was begun with the dust extract and improvement began after the second injection. Treatment was stopped in September, 1926. After the third treatment the patient has been free from asthma up to date (January 24, 1927).

Case IV.—L. E., an unmarried woman, thirty-eight years old, occupied as hotel clerk, was referred to me on October 15, 1926, with the following history:

She has had typical asthmatic attacks for the last seven years. Recently they have become very frequent, occurring daily and usually in late afternoon, and lasting one-half to one hour. About two or three times a year she experiences very severe seizures, lasting for several days and preceded by a three-day period of a "head cold." The first attack occurred in July, 1919. She has never had hay-fever, but has had hives for the last two weeks. There are no household pets and no plants in her home.

The patient had influenza in 1918. Except for the fact that her father had been subject to urticaria her family history is negative for allergy.

Examination showed a thin undernourished woman weighing 96 pounds. Temperature is 98° F., pulse 80, and respirations 24. The chest was thin and hyperresonant on percussion. Wheezing râles are heard. The heart showed no abnormal findings. There was tenderness of the large bowel on palpation. Cutaneous tests with all the stock proteins gave negative results, but the stock house-dust extract gave a positive reaction. An extract prepared from the dust from the patient's home also gave a strong positive reaction.

On October 26, 1926 treatment was begun, using the extract prepared from the dust collected from the patient's home. A slight attack of asthma occurred that night. On November 3, 1926 there was another attack of asthma. That was the last attack the patient has had. She has received twenty-six treatments and is still reporting for treatments at weekly intervals. Her chest is entirely clear at all times and she has gained 13 pounds.

Although it may be argued that in this instance the ultimate results are still open to question, the fact remains that by this method of treatment this individual has been entirely free from symptoms of asthma for nearly three months, whereas previously she was accustomed to have a daily seizure.

CLINIC OF DR. GRANT H. LAING

ST. LUKE'S HOSPITAL

MIGRAINE TYPES IN RELATION TO THE GASTRO-INTESTINAL TRACT

IT is not proposed to offer anything new as to migraine, but to bring out some of the rather interesting clinical features which present themselves as the result of some of its manifestations.

"It has been aptly said that since the attention of mankind is more or less chronically riveted upon its stomach, it is not unnatural to find this factor loom large in the history of migraine. Nearly all the sufferers from it will complain that they are bilious, in which word one recognizes the fact of previous generations, a direct descendent of the days when black bile was considered of so much importance in medicine."

The name migraine comes from hemicrania, one-sided headache, although not all migraine headaches are confined to one-half of the head, nor are all one-sided headaches migraine.

One might briefly outline a few of the more common theories as to the etiology of this extraordinary symptom complex which has been mentioned as "philosophically interesting though therapeutically pride breaking."

First, that it is a reflex from the eyes, ears, pelvis, etc. The oldest view is that of a simple reflex brought out by Tissot in 1813 in which he says that a sensory stimulation of any organ is reflected in the corresponding brain center.

Second, that it is a disorder of metabolism in which we find retention of toxic chemical substances. Although there are some reports in the literature of the urine and blood chemistry, a few of which show slight variations from the normal, the ultimate chemistry does not seem to have been worked out.

Third, that it is due to an endocrine dysfunction because of the association of the attacks with the menstrual periods, their cessation at the menopause (to which there are exceptions), and during pregnancy. Fisher, of London, feels that the periodic swelling accompanying functional overactivity of the pituitary is the best explanation. On the other hand, Erdheim, of Vienna, brought out the fact that there is a definite enlargement of the pituitary during pregnancy, and at times a hyperfunction as shown by the acute acromegalies which may occur during this period, subsiding with the termination of the same. Cushing has stated that pituitary tumors usually produce bilateral headaches not of this type.

Fourth, that it is due to a gastro-intestinal toxemia which is the result of dietetic errors. The advocates of this theory are not at all clear in their explanation of the mechanism concerned. If they mean the diarrheas which occur following the eating of irritating foods, it might be well to remember what Hurst has said about absorption being better from a liquid medium than a solid one. Thus the so-called toxic symptoms could be better explained on the basis of a diarrhea rather than on constipation.

Fifth, that it is a disease of the sympathetics producing vasomotor disturbances. Mollendorf described it as a paralysis of the sympathetics opposing the views of Dubois-Reymond, who thought of it as an irritation. This led to the recognition of two types, hemicrania-sympathetico-paralytica and hemicrania-sympathetico-tonica. The transient character of the focal symptoms of paresthesias and visual changes are all suggestive of vasomotor phenomena. On the other hand, in many cases no vasomotor symptoms are demonstrable.

Sixth, that it is an anaphylactic phenomenon. This has been emphasized by Pasteur Vallery-Radot, who says the symptoms are due to vagosympathetic disturbances and sums up the evidence from Bernard and Dubois-Reymond down to the present, concluding that the symptoms arise from vasoconstriction due to excitation of the sympathetic nervous system. Anaphylactic shock in animals is always associated with eosino-

philia, and Gansler reports eosinophilia of from 5 to 15 per cent. in 74 per cent. of migraines.

Seventh, that it is due to a process similar to that taking place in fatigue. The investigations of Weber, Mosso, and Bruno along these lines are noteworthy. They draw a parallel between fatigue and migraine: "Work causes a vasodilatory irritation of normal organs. This irritation, however, becomes vasoconstrictory in organs that are fatigued. Irritations of normal life cause the same vasoconstrictions in migraine patients as those brought about in the fatigued."

Buchanan has emphasized heredity and endeavors to prove that migraine tends to follow the mendelian law of inheritance. Viggo Christiansen, of Copenhagen, also agreeing with this, says, in addition, that he considers the disease as developing on some constitutional basis not related to an endocrine dysfunction, but manifesting itself in symptoms which are related to the sympathetic nervous system.

Richter, on the basis of 300 cases observed over a period of three years, produces evidence to show that local angiospasms occur.

The symptoms and course of a typical attack of only moderate severity might be described as follows: The patient may be awakened in the morning with a feeling of depression or fatigue, chilliness or dizziness, or even a "peculiar feeling in the back of the neck," as some patients describe it. Following this they may have certain ocular manifestations—specks, zig-zag lights, or other fortification—like phenomena or even hemianopsia. These sensory and ocular symptoms are soon followed by a headache, which starts out with a sensation of pressure in the temporal, supra-orbital, or orbital region, followed by actual pain, which, as the intensity increases, merges into nausea and vomiting. It must be remembered that each phase mentioned may be absent at times. The patient is very comfortable and usually active and efficient in the interim between attacks, and on questioning further one finds that the father, mother, sister, or brother in a great percentage of the cases is a sufferer from the above complaint.

With all the details being purposely eliminated to bring out certain clinical features, it is interesting to note the number of cases consulting the gastro-enterologist because of the patients, association of the nausea and vomiting with the gastro-intestinal tract, and the fact that in many instances they get relief from a bowel movement usually the result of a cathartic which they have taken at the onset of the prodromal symptoms.

One hundred and forty-five cases with headaches of the migraine type (in varying degrees), that is, with visual disturbances, headaches, nausea and vomiting, consulting an internist were reviewed.

Abdominal discomfort other than the nausea and vomiting associated with the headache was complained of in 121 cases. This discomfort was described all the way from gas pressure or fulness to heaviness and cramp-like distress.

Constipation.—This symptom was complained of in 131 cases, and the great majority of these were taking cathartics or large enemas.

Nausea with the other disturbances but without the vomiting was present in 9 cases.

Hemianopsia was present in only 2 cases as far as could be determined.

Sex.—One hundred and sixteen cases were found in females and 29 in males.

Heredity.—Thirty-one cases out of 145 gave no family history or did not know. Of the remaining 114, the mother and grandmother had similar headaches in 69 cases, the sister in 32, the father in 9, and the brother in 4.

The chief complaints were:

1. Abdominal distress which was usually associated with constipation and was becoming progressively worse.

2. The headaches were increasing in frequency and severity, from one a month to three or four a month. In less than one-fourth of the cases did the patient mention the headaches as the predominant complaint.

Since the patients associate the headaches with the "stomach" because of the above symptoms and because certain foods

"disagree" with them, they start limiting their diet usually to the non-residual foods, and, in turn, develop constipation which the physician realizes is due in large part to the character of the food. The patient trying to avoid constipation starts catharsis.

We know that under normal conditions the motor processes of the gastro-intestinal tract which is under the control of the autonomic nervous system proceed without being registered in the consciousness of the individual. When this normal rhythm is interrupted, however, he becomes aware of certain disturbances for which there may be a double explanation. First, we usually have an individual whose sympathetic nervous system is more sensitive to outside stimuli, and second, we have an irritation of the tract itself. With the added factor of abdominal discomfort becoming progressively worse, and the worry attached thereto, we find the headaches increasing in frequency and severity.

With this résumé of the important features of this symptom-complex, I shall present to you a patient with typical history.

Miss A. V., aged twenty-one, came in complaining of "stomach trouble," headaches, nausea and vomiting, constipation severe enough to require catharsis, and nervousness.

The "stomach trouble," which has been present for the last two years, consisted of generalized abdominal distress relieved by a bowel movement. Constipation was marked and was present for the same period of time. Headaches of the migraine type have been present since puberty.

Physical examination was essentially negative except that the patient was underweight and the colon was tender to pressure over its course and spastic along the descending portion.

We recognize this general abdominal distress as referable to the large bowel.

Assuming as correct the hypothesis that migraines are mostly vasomotor neuroses, we have a foundation on which to work. It is essential then to look for all the peripheral causes which may be factors in upsetting the balance of vascular control held by the sympathetic nervous system.

With this in mind we can now turn to the treatment carried out in this patient. The abdominal distress had been brought on for the most part by the use of purgatives or large enemas which she had been in the habit of using in an attempt to alleviate the condition. Certain foods as members of the cabbage family, apples, etc., containing organic acids (in addition to cellulose), which are irritant or cathartic in nature, have, together with the catharsis, aggravated the patient's colon, bringing about perverted contractions.

The cathartics were stopped and a strict dietary régime was not instituted except to eliminate a few of the fruits and vegetables which are chemically more irritating. She was taught that sufficient residue in a diet is the main essential to secure normal bowel movements. Small doses of bromids and some form of salicylate were given over a short period of time.

She has been under treatment for the past six months, and today reports that the abdominal distress has practically disappeared and has had only three headaches, whereas before beginning treatment she had two to three a month.

The same line of treatment was employed in the 145 cases cited above, and the incidence and severity of the headaches have been markedly decreased in 80 per cent. of the cases.

CLINIC OF DR. E. F. FOLEY

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ALCOHOLIC CIRRHOSIS OF THE LIVER

THE chronic use of alcohol as a beverage is attended with well-recognized clinical and pathologic effects on the liver. There is a marked dissimilarity between the early and late changes, and these changes produce well-known characteristic symptoms. The early symptoms are well illustrated by this case. This man is forty-five years of age, a mechanic by trade, who entered the hospital with these complaints: Insomnia, anorexia, loss of weight, jaundice, nausea, but no vomiting. He states that he was perfectly well until four months ago, when he began to drink following some financial difficulties. At onset a pint of moonshine would last three days, but he gradually increased the amount, so that at the end of a month he was taking a pint a day. At that time he discontinued his work as a mechanic, and turned his attention more seriously to his imbibing. He manufactured a still in his basement for the production of moonshine and likewise began brewing beer. For the past three months he has been constantly under the influence of liquor. He estimates that he drank about 1½ pints of "moonshine" a day and about a quart of beer. He further states that he ate only one meal a day, a large meal in the evening. In the morning he could not eat breakfast because he was nauseated and, to correct this nausea, he took an "eye opener." About a week ago his wife noticed his eyes were yellow and a few days later he noticed that his skin was yellow and his urine highly colored.

Past History.—Had usual childhood diseases. Influenza in 1918. No operations or accidents. Venereal disease denied.

Family History.—Negative.

Marital History.—Married twenty years. Four children. Wife had no miscarriages.

Physical Examination.—He is a poorly nourished white male. Temperature 99.2° F. Respiration 20. Pulse 90. The skin is moist, and is definitely jaundiced, as are the scleræ and soft palate. There are no ocular palsies, and pupils are equal, round, and react promptly to light and accommodation. The teethare carious and there is some pyorrhea. The tongue is heavily coated, tremulous, and protrudes in midline. The heart and lungs are negative for any gross pathology. The abdomen is soft, not distended, and there is no shifting dulness in the flanks. The liver is palpable, the edge can be felt about four finger-breadths below the costal margin. The palpable surface is smooth, the edge is rounded, and not particularly tender. The spleen is not palpable. There are no palpable masses in the abdomen. There are no paralysis or deformities of the extremities, but there is a coarse tremor of the fingers. The reflexes are all lively, but equal on both sides. There are no pathologic reflexes present. Urine: Specific gravity 1024, acid, no albumin, no sugar, acetone or diacetic acid. There are no casts, pus, or red blood-cells. The urine gives a positive test for bile. White count 10,200. Hemoglobin 85 per cent. Red blood-cells 4,100,000.

Spinal fluid, normal pressure, Pandy negative, 1 cell per cmm. Wassermann negative, blood Wassermann negative.

The dye test for liver function, using bromsulphalin, shows a retention of 40 per cent. in one-half hour. This amount of retention of the dye indicates considerable impairment of the ability of the liver to remove this foreign substance and suggesting a rather extensive lesion. The icterus index is 38, indicating a moderate jaundice. The van den Bergh is strongly positive in the first phase and the serum bilirubin measures 4.8 mg. per cent., indicating an obstructive type of jaundice of moderate degree.

To summarize, there is a history of excessive alcoholism of recent date, and the findings of enlarged liver and jaundice in

a rather acutely ill individual. The enlargement of the liver, of course, can be attributed to the effect of the alcohol. The histologic changes that occur as described by Mallory[1] are characterized by a necrosis of the liver cells, commonly in region of the portal vessels, followed by a leukocyticr eaction which brings about the disappearance of the necrotic cells. Along with these degenerative changes there is active regeneration of liver cells and proliferation of connective tissue and a fatty infiltration, all of which are grossly manifested by the huge liver, which is commonly fatty. At this point death not infrequently occurs and the chief finding at necropsy being the large fatty liver. A series of such cases have been described by LeCount and Singer,[2] and they believe the cause of death to be due to replacement of the glycogen in the liver cells with fat. As the condition progresses the connective tissue contracts and in contracting occludes bile-ducts and blood-vessels throughout the liver. The development of jaundice depends on the regularity of the process throughout the liver; its occurrence clinically is very irregular. The presence of jaundice cannot be used as an index of severity of parenchymal damage, for it occurred in only one of LeCount and Singer's fatal cases. Hence the Van den Bergh and icterus index are of no prognostic value in this type of case, for they are not concerned with the vital function, the failure of which causes death in these cases.

This man is still in a precarious condition, but his prognosis gets better each day, for he has been removed from the cause of his trouble and being fed at regular intervals.

If he overcomes this acute episode and subsequently relapses into his former mode of living in a few years we expect him to present a picture similar to this second case.

The other case is a woman. One year ago she was well, then she noticed her abdomen was becoming larger, but thought she was getting fat because she was not wearing a corset. She noticed, however, that she was not putting on any flesh elsewhere. She now began to complain of nausea in the morning before breakfast which was continued until she ate. She noticed that her shoes were too tight in the afternoon, but in the morning

the shoes were loose. This continued, and gradually became worse until they were swollen even in the morning. She then went to bed during the day, and elevating the feet the rest of the day would cause the feet to become less swollen, but increase the swelling in the abdomen. In August, 1926 the swelling of the abdomen became so great and the pain from the tightness of the abdominal wall increased to such a degree that a doctor was called, who tapped her and withdrew 3 quarts of fluid. She felt much better and the swelling decreased in legs, but gradually the abdomen began swelling as before until she became so uncomfortable that she entered the hospital. Since she has been in the hospital she has been tapped twice, with the removal of 9000 and 7500 c.c. She complained also of dyspnea and palpitation on exertion, and she volunteered the information that she thought the fluid was due to failure of the kidneys because she passed her urine so infrequently.

Medical History.—Mumps. Chickenpox. No diseases of adult life. Surgical denied. Venereal disease denied.

Family History.—Mother died of old age. Father died of cholera. One brother died of asthma. Two sisters living and well. Husband sixty-five years old. No children. No tuberculosis and cancer in family.

Habits.—Appetite poor. Nauseated in morning. Bowels regular. Drinks wine and makes own home-brew. Formerly drank beer very heavily. No tobacco, tea, or coffee.

Examination revealed very poorly nourished white female about fifty years of age. Pupils unequal, round, react sluggishly to light and accommodation. Mouth and nose negative. Chest and neck negative.

Vaginal: Negative except for senile changes.

Abdomen: Protuberant frog belly type, fluid wave present. Distended superficial veins. Skin of the abdomen shiny and tense.

Liver, spleen, and kidneys not palpable. Following paracentesis liver palpable. Dulness in flanks, which shifts as patient changes position. Edema of ankles and legs.

Reflexes normal and equal.

Urine: Pale, straw color, acid, specific gravity 1020. No albumin, sugar, acetone, diacetic acid, indican, or bile. No casts or red blood- or pus-cells.

Blood: Sugar 86 mgm. of 100 c.c. blood. Non-protein nitrogen, 36 mgm. for 100 c.c.; hemoglobin, 56 per cent.; red blood-cells, 3,040,000; white blood-cells, 4500.

Differential count: Polymorphonuclear leukocytes, 56 per cent.; lymphocytes, 22; mononuclears, 19; eosinophils, 3.

Blood Wassermann negative.

Fluid from peritoneal cavity. Amount, 9000 c.c.; specific gravity, 1008; slight amount of albumin; few degenerated leukocytes; Rivalta test negative; Wassermann negative on ascitic fluid.

Smears and cultures negative.

Guinea-pig inoculated was negative for tuberculosis cultures.

Blood-pressure 144/60.

Bromsulphalin 25 per cent. retention of dye in thirty minutes.

Icterus index 8.

Van den Bergh—direct negative.

Serum biliribum 2 mg. per cent.

Temperature 98.6° F.

Pulse 72.

Respiration 20.

x-Ray of gastro-intestinal tract is negative for organic lesion.

x-Ray of the chest showed high diaphragm apparently from pressure below.

Diagnosis: Alcoholic cirrhosis.

The diagnosis rests largely on the determination of the cause of the ascites. With the history of heavy use of alcohol we will be prejudiced toward a diagnosis of cirrhosis of the liver. A failing myocardium usually produces edema about the ankles at first and the enlargement of the abdomen later. In this case the first symptom the patient noticed was the enlargement of the abdomen and the disappearance of the edema when the fluid was removed from her abdomen to recur in her ankles after the abdomen again became large.

The examination of the ascitic fluid in this case shows it definitely to be a transudate excluding any chronic peritonitis associated with ascites such as tuberculosis.

The dilation of the veins of the abdominal wall indicates the establishing of a collateral circulation between the epigastric and internal mammary veins and suggests a portal obstruction. In a negative way there is the absence of blood findings of the blood dyscrasias. Lues is excluded by the absence of other signs of lues, by the course of the disease, by the ascites and by the negative Wassermann on the blood and ascitic fluid. There is no evidence of a primary carcinoma in the neighboring organs to give rise to metastases, the duration, the absence of jaundice, all speak against malignancy.

The dye test to which reference will be made later indicates the impairment of function such as would be present in a rather extensive lesion, and in this type of case is of more value than the tests for impairment of biliribum metabolism, for here at least they do not suggest the amount of damage which we have reason to suspect and which is indicated by the dye test.

Alcohol apparently causes fatty changes so that the early alcoholic liver is the fatty liver associated with areas of necrosis and proliferation of connective tissue. The sclerotic changes which occur to produce cirrhosis are probably the result of inflammatory reactions resulting from intestinal infection and localizing in the areas of damaged liver cells. The ascites may be the result of several factors, the most probable being that it is mechanical in origin. Compression of the vessels by contraction of the connective tissue as the disease progresses offers an increased resistance to the blood flow[3]; or, as has been suggested by Herrick,[4] establishment of a more direct communication of the arterial pressure to the portal blood through the dilated vessels.

The symptoms of which this patient complains chiefly are the result of the pressure from the fluid in the abdomen. The pain and tightness of the abdomen which led her to seek medical aid are the result of stretching and tearing of the skin of the abdominal wall. The very weight of the fluid—at one paracentesis 9000 c.c. were removed which weighed approximately 18 pounds

—causes a considerable degree of discomfort. The edema of the ankles, resulting from pressure on the vena cava and iliacs, the palpitation and dyspnea from pressure on diaphragm are all mechanical.

The establishment of the collateral circulation leads to the development of esophageal varices, and to a chronic passive congestion of the stomach and bowel. Primarily the collateral circulation is protective in nature, but as the disease progresses and more and more blood is shunted away from the liver the effect approaches that of an Eck fistula and consequently there is a gradually increasing toxemia.

In the course of the disease the appearance of ascites is a bad prognostic sign, repeated tapping becomes necessary with gradually increasing frequency, and even though this fluid possesses a high degree of resistance to infection, this danger is always present.

Since the introduction of bromsulphalin by Rosenthal and White,[5] we have used it in about 200 cases instead of phenoltetrachlorphthalein. In none of the cases was the administration of bromsulphalin attended by nausea or vomiting, or have we seen any local irritation or phlebitis from it. These reactions, both general and local, occur not infrequently following phenoltetrachlorphthalein and practically condemn its use. In addition, it is necessary to withdraw only one sample of blood, usually one-half hour after the administration of the dye, instead of two or three, as with the older dye, a point that is appreciated by the patient. It does not require as much time and is simpler. Besides this the advantages claimed for it by Rosenthal and White[5] are that: (1) It is specific, and when properly performed abnormal results mean disturbed function. (2) Easy to perform and free from error. (3) It affords a quantitative measure of the severity of impaired function.

It has invariably been positive in cases of obvious liver disease, the retentions vary from 10 to 100 per cent., and it has not been falsely positive in any case of suspected liver disease that has come to postmortem. Clinically, then, its chief value lies in detecting the presence of liver disease, but, like all other similar tests, in its interpretation it should be considered as a part of

the evidence and not all of the evidence in making the final diagnosis.

In all cases where the retention has been over 85 per cent. death has ensued within a few days. The reading of, say, 50 per cent. does not mean, necessarily, that anatomically there is only 50 per cent. of liver tissue, but that this particular function of removing a specific dye from the blood-serum is impaired 50 per cent.

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CLINIC OF DR. HARRY A. SINGER

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WALLED-OFF AND FREE PERFORATION OF PEPTIC ULCER

IN order to treat a medical or surgical case successfully a knowledge of the nature of the underlying process and of its manifestations is of paramount importance. This is particularly true in connection with the management of peptic ulcer and its complications. The pathology and symptomatology of uncomplicated ulcer being quite well understood, this condition presents to the attending physician in regard to diagnosis and treatment no immediate problem. The morbid changes and symptoms which characterize acute perforation of a peptic ulcer into the free abdominal cavity and the effects resulting thereform are likewise sufficiently well known to render the diagnosis and therapeutic procedures quite clear.

Occupying a position between the uncomplicated and the acute, free, perforated ulcer is a type which although ushered in by violent symptoms of a ruptured viscus, fails to develop evidences of a diffuse peritonitis. The nature of this type of case is in general but poorly understood, with the result that lack of recognition and mismanagement as a rule follows. I refer to a group of ulcer cases described by Moynihan and by Lund under the heading of "Subacute Perforation," and by Schnitzler as "Gedeckte Perforation." From a number of such cases with walled-off perforations which have been treated during the past year at the Research and the Cook County Hospitals and which are examined periodically in the out-patient department the following 3 are chosen. These particular cases are selected for

the purpose of furnishing the reader with not only a clear conception of the affection but also with a picture of the more frequent clinical variations.

Case I.—H. W., male, forty-four years of age, admitted October 2, 1926. The patient stated that he had been suffering during the past two years periodically from attacks of "sour stomach." The attacks which lasted from two to three months manifested the usual characteristics of ulcer, consisting mainly of burning coming on regularly after meals and relieved by milk or soda. The present attack of "sour stomach," which was similar to but more intense than previous attacks, began two days prior to entrance. Thirteen hours before admission following the ingestion of a large amount of food and alcohol the patient experienced severe knife-like pain above the umbilicus which caused him to double up and roll about in agony. The pain was maintained in paroxysms which occurred every few minutes. Seven hours after the initial severe pain the patient vomited without amelioration of symptoms. He complained of some difficulty in respiration. When examined he was found to have a subnormal temperature (98° F. rectally), the pulse rate was 110, and the respiratory rate 44. It was recorded that the patient appeared to be in shock for the extremities were cold and clammy and the pulse was almost imperceptible. Over the right rectus there was felt some increase in resistance, whereas in the epigastric region and also in the right upper quadrant tenderness was elicited. The liver dulness occupied normal bounds. The white blood-count was 10,400. The fluoroscopic examination showed no free air in the peritoneal cavity. The patient who responded almost instantaneously to intravenous saline, external heat, and stimulants was treated expectantly since his condition was thought to be the result of a walled-off, perforated peptic ulcer.

The following morning, twenty-four hours after the acute onset, the aspect of the case had changed entirely. The patient appeared quite comfortable and offered no complaints. The rigidity and tenderness of the upper abdomen were entirely absent. The pulse rate was normal, as was the temperature, but

the respiratory rate was still somewhat increased. The white blood-count had not altered. The fluoroscopic examination again failed to reveal the presence of escaped air from the stomach. No further symptoms appeared and the patient was placed on Sippy management for ulcer. At the expiration of two weeks examination undertaken with the aid of a contrast meal showed the presence of a defective bulb. The patient now returns for observation and states he has felt entirely well since the acute attack subsided.

Case II.—B. F., female, thirty-five years of age, had been previously treated in the hospital on the basis of a classical history, persistent occult blood in the stools, and positive *x*-ray findings for duodenal ulcer. She was last seen on January 6, 1927, at the follow-up clinic, when she admitted that she was not observing her dietary instructions strictly. On January 10th, at 9 P. M., four hours following a full, mixed meal, she experienced severe pain in the back, which was soon referred also to the epigastrium, where it localized and became excruciating and intolerable. She vomited repeatedly. Upon readmission to the hospital, five hours after the initial pain was experienced, she was given three Sippy powders which she immediately vomited. The temperatre was 98° F., the respiratory rate 28, and the pulse 80. No cold sweat was noted. The upper abdomen was tender upon deep pressure and only slightly rigid over the right epigastrium. The leukocyte count upon entrance was 16,000. No air was seen in the peritoneal cavity upon fluoroscopic examination, nor was there any diminution upon percussion in the area of liver dulness. The following day the patient felt greatly improved, but had a temperature of 100° F. and a pulse rate of 116. The abdominal findings still persisted. There was no diminution in the peristaltic sounds nor had the patient vomited since entrance, and on the third day, being completely relieved, the patient resumed ulcer treatment. Frequent examinations made throughout the course failed to reveal further abdominal findings or any accumulation of air in the peritoneal cavity. Repeated white counts showed a gradual diminution in the num-

ber of leukocytes (10,800 on the third day). The patient now returns to the follow-up clinic, stating that she had a similar attack from which she recovered in two days accompanying her last menstrual period. She has noted for several months a time relationship between her menses and exacerbations of her ulcer symptoms.

Case III.—L. C., female, fifty-two years of age, who suffered from ulcer symptoms for four years, and had a gastro-enterostomy performed three years prior to entrance, complained of pain which was similar to her previous ulcer distress except for an increase in severity and lack of response to the usual doses of alkali. These symptoms had been present for eight days previous to entrance accompanied by frequent vomiting of sour, watery material. While under observation the patient developed severe cramp-like pains in the upper abdomen followed by signs of collapse. The pulse became rapid and weak, respirations shallow, and a cold sweat appeared on the patient's brow. Vomiting occurred at every attempt to ingest food or alkalies. Upon physical examination there was found a localized point of superficial tenderness slightly to the left of the midline with little or no rigidity of the abdominal muscles. There was no obliteration of liver dulness, nor was there evidence of free air in the peritoneal cavity as determined with the aid of the fluoroscope. The temperature was 98° F., the pulse rate 124, and the respiratory rate 24. The diagnosis of a walled-off perforation was made and sedatives administered. The following morning the patient was quite comfortable, sitting up in bed, and except for occasional and slight abdominal distress had no complaint. Rectal alimentation was instituted, and as soon as it was thought safe, *x*-ray examination with a barium meal was undertaken and a gastrojejunal ulcer demonstrated. Operation performed December 29, 1926 (Dr. Karl A. Meyer) disclosed a gastrojejunal defect which had become densely adherent to the anterior abdominal wall which formed the ulcer base. No evidence of the previous ulcer was found at operation. The old gastro-enterostomy was undone and the ulcer area removed. The patient now returns for observa-

tion and follow-up treatment. The last *x*-ray showed in addition to an irregularity at the site of operation a deformity of the duodenum, presumably due to scarring rather than to an active ulcer, as the stools have been persistently negative for occult blood and the patient is entirely symptom free. She reports at this visit no discomfort and presents a steady increase in weight.

DISCUSSION

Perusal of the foregoing cases forces one to the conclusion that the underlying process is essentially different from that of free perforation. In the latter the attack of pain is followed by increasing collapse (except perhaps for a short euphoric intermission) and the supervention of a classical picture of diffuse peritonitis with its almost inevitable fatal outcome. In the exceptional case in which recovery does occur there ensues following the usual signs of rupture a prolonged and stormy convalescence with gradual abatement of the symptoms and findings of a wide-spread peritonitis. It is noteworthy that in all 3 aforementioned cases, although at the outset the symptoms were those generally ascribed to free perforation, almost complete recovery occurred within a day or so. Furthermore, in not one was there any evidence whatsoever of the development of acute peritonitis or abscess. A consideration of the nature of the morbid changes in the so-called walled-off or subacute perforation elucidates clearly the basis for the difference in behavior between this type and free rupture into the abdominal cavity.

Pathology.—In place of the establishment between the stomach or duodenum and the general peritoneal cavity of a communication which allows the escape of unrestricted quantities of material, such as occurs in free rupture, there develops at the site of perforation a process which prevents or limits the gastroduodenal leakage. As a result the peritonitis which develops is entirely local and readily becomes circumscribed. It is, therefore, the extent and degree of peritoneal soiling that constitutes the essential difference between the walled-off and free perforations. Since the severity of peritonitis following perforation is proportionate, other things being equal, to the amount and di-

tribution of the escaped gastro-intestinal content, it is quite apparent that in dealing with a readily sealed or walled-off perforation little or no peritoneal inflammation will result. This prevention or limitation of leakage into the general peritoneal cavity fully explains the failure of diffuse peritonitis to develop and likewise accounts for the rapid recovery seen in the cases of "protected" perforation.

What are the factors which lead to the limitation of the escape into the general peritoneal cavity of gas and fluid? Schnitzler attributes the absence of a diffuse peritonitis to the circumstance that the lesion readily becomes covered (gedeckt) by an adjacent structure. In 4 cases in which he performed laparotomy within a day or two after the patient had recovered from the acute symptoms, he was able to demonstrate fibrinous adhesions between the site of perforation and the under surface of the liver, the pancreas and the anterior abdominal wall respectively, completely covering and effectively plugging the point of rupture. In addition to the covering of the base of the perforating ulcer by a viscus or the abdominal wall Moynihan gives as causes of restricted leakage the following: 1. An empty condition of the stomach at the time of rupture. 2. The presence of layers of protective exudate which are laid down as the ulcer deepens, so that when final dissolution of the wall occurs there is already a barrier almost or entirely impenetrable. (The term "subacute" perforation used by Moynihan is particularly applicable to this group, since it is in these cases that the less stormy onset is encountered.) 3. Plugging of the opening in the ulcer with a tag of omentum. To the aforementioned conditions which favor checking of the escape of gastro-intestinal contents may be added the occurrence of a minute perforation through which the leakage is sufficiently impeded to permit circumscription. It is quite conceivable that a piece of undigested food may act as a valve in these pin-point or larger openings for a sufficient length of time to permit walling off.

Symptomatology.—The manifestations in the classical case of walled-off ulcer may be divided into three stages. The first stage occupies a number of months or years during which time the pa-

tient suffers from periodic attacks of ulcer distress. The character of this distress, such as its relationship to food taking, the effect of alkalies, etc., are too well known to warrant enumeration here. During the second stage an aggravation of the previous symptoms supervene. The relatively mild epigastric discomfort is replaced by a more intense, persistent, and refractory sensation which is described as actual pain. The pain now appears within a shorter interval after eating or after the ingestion of powders, and is, in general, less responsive to the usual measures which previously afforded relief. Vomiting becomes more frequent and produces but temporary alleviation of pain. These inaugural symptoms presumably due to active penetration of the ulcer continue for a variable period of time (from one to several days), sometimes increasing in intensity until the dramatic manifestations of the third stage due to actual perforation make their appearance. In this latter stage the patient is seized with violent and intolerable pain in the epigastrium which causes the victim to double up. Accompanying the agonizing pain are symptoms of prostration and in some cases evidences of collapse. Vomiting usually occurs shortly after the onset of the pain and may be oft repeated. No relief is obtained as the result of emptying the stomach, nor are alkalies, even if retained, effective in even mitigating the suffering. As a rule, the pain although severe is less intense and the signs of collapse are less obtrusive than in cases of free perforation, but not necessarily so.

In not all cases are three stages represented, for it frequently happens that the premonitory symptoms of the second stage are missed. These manifestations of impending perforation occur characteristically in the "subacute" perforations and are often entirely absent in the acute walled-off cases. In a number of instances the ulcer remains latent for several weeks or months, and without any warning or premonition the patient is suddenly seized with violent pain. It is quite exceptional for acute symptoms to develop without a story of previous ulcer distress if careful inquiry is made.

Although the symptoms at the outset and during the first few hours are indistinguishable from those of free perforation,

except perhaps on the basis of degree of severity, the physical findings differ sufficiently to permit discrimination between the two types. The steadfast, board-like rigidity of the upper or entire abdomen so characteristic of acute rupture of a peptic ulcer into the free peritoneal cavity is likely to be absent in the walled-off perforation. One finds here, as a rule, only a localized area of increased resistance which is frequently unilateral and remitting. When the ulcer perforates posteriorly the anterior abdominal wall may remain quite supple. Tenderness is readily elicited and corresponds in location to the site of perforation. The degree of pressure required to provoke the pain is dependent to a great extent upon the location of the ulcer. A paravertebral dorsal pain-point can frequently be demonstrated. Upon percussion the liver dulness is found to be preserved notwithstanding the circumstance that the stomach may contain a large quantity of gas. If auscultation be applied to the abdomen, peristaltic sounds will be heard normally, especially over the lower two-thirds. The absence of free air in the peritoneal cavity in the presence of gas within the stomach is a highly significant finding, for it indicates in general a plugged or covered perforation. The leukocyte count at the early stage is normal or slightly elevated. The temperature is usually subfebrile, but may rise slightly above 100° F. The respiratory rate is, as a rule, increased, the excursions being limited and thoracic in type. Dyspnea, in fact, may be quite pronounced. The pulse is increased in rate, small, and easily compressible; the blood-pressure usually falls. Operation undertaken within the first twenty-four hours will disclose one of the conditions described under the heading of Pathology. The fibrinous adhesions at this stage are readily separated and when torn apart do not cause bleeding.

In the cases in which surgery is not resorted to, after the lapse of several hours to a day the clinical picture undergoes in many cases a most remarkable change. Instead of the critically ill patient seen at the time of the first visit, one finds an individual who from external appearances shows no evidence of discomfort or sickness. As a matter of fact, in the third case presented today I failed to recognize the patient when I saw her for the

second time and could hardly believe it was the same individual, so striking was the transformation. Physical examination at this stage is practically negative except for slight tenderness in the epigastrium, and, according to Schnitzler, over McBurney's point also. The tenderness in the right lower quadrant is explained by the escape of a limited quantity of fluid which gravitates along the inner border of the ascending colon and collects in the right iliac fossa. The temperature drops, the pulse and respiratory rates become normal or nearly so, while the leukocyte count may remain slightly elevated, but does not rise over above that of the first few hours. Roentgenographic examination persists in indicating the absence of a gas-bubble. Laparotomy is not infrequently performed at this stage, when the base of the ulcer is generally found covered by an adjacent structure to which it is united by readily removed fibrinous adhesions. As time progresses the fibrin becomes transformed into firm fibrous tissue which prevents separation without instrumental division. If surgical intervention is not carried out the subsequent course is that of a penetrating ulcer with its well-known symptoms and signs.

As might be anticipated, in not all cases is the change from the initial violent illness to that of comparative comfort an abrupt and permanent one. It is to be expected that where the walling-off process does not become effective until after the escape of some gastro-intestinal content, signs of local peritonitis will be maintained until absorption occurs. Where a moderate amount of gastro-intestinal content escapes and becomes well circumscribed the patient's symptoms, although far milder, persist for a few days, after which time, if the customary medical ulcer regimen is carried out, practically complete recovery from the acute illness follows. The formation of a true perigastric or periduodenal abscess associated with signs of sepsis, although discussed more frequently and described at greater length than sealed or walled-off perforation, is of far less importance from the standpoint of frequency. However, its occurrence is not to be lost sight of, and whenever symptoms pointing to a walled-off perforation are followed by persistent fever and its concomitants

the presence of an abscess should be suspected and a palpable mass sought for in the epigastrium. Another sequel of walled-off perforation, though actually of rare occurrence, is a secondary separation of the fibrinous adhesions. It is quite conceivable that a plugged or sealed perforation may subsequently become reopened and lead to further peritoneal soiling. As a result there may ensue a diffuse peritonitis the symptoms and signs of which should be looked for daily in all cases with an antecedent history suggestive of recent walled-off perforation.

Diagnosis.—As pointed out by Boas, the diagnosis of a walled-off perforation is beset with many difficulties. However, as a result of extensive study of its manifestations, the condition is becoming more frequently recognized. The symptomatology as outlined above is sufficiently distinctive to permit a correct diagnosis in a fair proportion of cases. The affection which it most closely resembles is the free rupture into the peritoneal cavity not only at the outset but also after the alarming symptoms have subsided. There are a number of differences, however, which render a distinction between these two similar conditions possible in the great majority of cases. The aggravation of ordinary ulcer symptoms just preceding actual rupture speaks for a relatively slow and, therefore, readily walled-off perforation. This is particularly emphasized in the term employed by Moynihan (subacute) to describe those perforations which do not lead to diffuse peritonitis. However, the premonitory symptoms occur with sufficient frequency preceding rupture into the free abdominal cavity also to render this point of only relative value in the differentiation. More important is the severity of the symptoms, for although the pain in the sealed perforation is frequently intolerable, it is not as intense, as a rule, as is free rupture, nor are the symptoms of collapse as frequent or as marked in the walled-off cases. The physical findings in the protected perforation are localized, inconstant, and rather unobtrusive. The rigidity here is localized and not maintained, whereas in the type with free rupture an unremitting, board-like resistance involving both recti is practically always found. The obliteration of liver dulness when not due to tympanites

indicates escape of air into the general peritoneal cavity and should in itself make the distinction. Pain referred to the shoulder speaks for acute perforation, as also does tenderness in the cul-de-sac of Douglas. The white cell count is of relative importance in that in free perforation a more pronounced leukocytosis is noted. Of particular value in the differential diagnosis is the presence of an air bubble recognized upon fluoroscopic examination as a narrow, sickle-shaped, transulcent zone which alters location with change in position of the patient. (For details regarding the *x*-ray findings in cases of free perforation see Vaughan and Brams.)

When the patient is seen later in the course of the illness, that is, after the severe symptoms have subsided, it is likewise essential to differentiate the walled-off from the free perforation. It is of paramount importance to bear in mind the fact that in cases of acute perforation which are followed by diffuse peritonitis a euphoric intermission (period of repose, Symonds), which gives both the patient and physician a false sense of security, is not infrequently encountered. This intermission corresponds in general to the quiescent period which follows the development of gangrene or rupture of an appendix. The pain ceases and the patient passes into a state of comparative comfort which is especially enhanced by the administration of opiates. Misinterpretation in this stage leads, as a rule, to disastrous results. Notwithstanding the circumstance that a patient with an acute free perforation during this period of repose is fairly comfortable, it will be found that the rigidity of the abdomen, although not as marked as at the outset, still persists, and that some degree of tympanites has supervened. Tenderness is found to be rather diffuse in contradistinction to the walled-off group where the tenderness is limited to the epigastrium and possibly also the right iliac fossa. The temperature is still elevated, whereas the leukocyte count, a finding of extreme importance, is higher than during any time previously. If during the initial stage pneumoperitoneum was not determined to be present and the patient instructed to lie upon the left side in the interim, repeated fluoroscopic examination usually shows the presence of

demonstrable air in the peritoneal cavity if the perforation is not protected.

The following case, which depicts clearly the clinical picture and findings of acute rupture of a peptic ulcer into the free abdominal cavity, is presented for comparison with the previous 3 cases of walled-off perforation. F. C., male, thirty-two years of age, was admitted August 6, 1926, with a history of ulcer symptoms, occurring periodically for the past year. On two or three occasions he had noted tarry stools. Three hours before entrance the patient was suddenly seized by an agonizing pain which caused him to double up and to restrict his respirations. Pain was at first referred to the epigastrium, but later became diffuse. No distinct inaugural symptoms preceded the attack. Vomiting occurred previous to entrance and was repeated shortly after admission to the hospital. Upon physical examination the patient was found to present the typical board-like rigidity and there was diffuse tenderness over the entire abdomen, particularly marked in the epigastrium and the right iliac fossa. Upon percussion partial obliteration of liver dulness was noted. The white blood-count was recorded as 29,300. En route to the operating room the patient was fluoroscoped and an air bubble between the diaphragm and the liver signifying free gas in the peritoneal cavity was seen (Dr. C. A. Matthews). At the operation (D. Karl A. Meyer) a perforation of the stomach through which air and gastric contents were escaping was found communicating with the abdominal cavity.

The other conditions with which walled-off perforation of a peptic ulcer is confounded are, in general, those diseases which are considered of importance in the differential diagnosis of rupture into the free abdominal cavity of a gastric or duodenal ulcer. The conditions which are most frequently mistaken for ruptured ulcer are dealt with so extensively in surgical treatises that a detailed account of them is hardly warranted at this time. However, a few general statements which are pertinent may prove of value.

It is well to keep foremost in mind the fact that in practically all cases of walled-off perforation an antecedent history of the

typical peptic ulcer syndrome can be elicited. In the differentiation of cholecystitis (acute exacerbation) or cholelithiasis particular care must be exercised in evaluating the anamnesis, for many cases of cholecystitis give a history of epigastric distress after meals. The discomfort is likely, however, to follow certain articles of diet, particularly (selective dyspepsia) such as greasy and fried foods and roasts. The epigastric distress is more frequently described as a sensation of pressure often associated with flatulence, the latter, as a rule, relieved by belching. If alkalies are resorted to, unless belching is induced, little or no relief is afforded the patient. Belching is frequently an outstanding feature in gall-bladder disease and should be inquired into. The habitus and sex of the individual are also of aid in the differentiation, for generally speaking biliary disease afflicts with predilection obese females, whereas thin males are predisposed to ulcer. When the cecum is undescended and the appendix occupies a high position in the abdomen or is situated near the midline difficulties in diagnosis frequently arise. Of particular importance is the fact that acute appendicitis does not present the stormy onset which characterizes the walled-off perforation. If the patient is seen for the first time in the later stages the diagnosis, as pointed out by Schnitzler, of a receding appendicitis is frequently made on account of the residual tenderness in the right iliac fossa. A careful history, however, will obviate, as a rule, the possibility of error.

Acute pancreatic disease (pancreatic fat necrosis, pancreatitis, and apoplexy of the pancreas), although of relatively infrequent occurrence, resembles the walled-off perforation of a peptic ulcer so closely as to merit special consideration. The pain in acute disease of the pancreas is far more intense than in walled-off perforation, whereas the degree of collapse is often alarming. I recently saw a stout woman apparently in good health who was in the hospital under observation for gall-bladder disease die within a few hours after the onset of overwhelming pain and signs of extreme collapse. At the postmortem a chronic cholecystitis with cholelithiasis and hemorrhagic necrosis of the pancreas were found. The pain is usually transverse, extending

across the upper abdomen. As in gall-bladder disease, the history, sex, and habitus of the patient are to be taken into account. Not infrequently icterus occurs. The finding of sugar in the urine and a diminution in the pancreatic ferments of the stool when available speak for pancreatic involvement.

Mesenteric embolism is another condition to be considered not only from the standpoint of suddenness of onset and severity of pain but also on account of the melena which occurs in this condition. A tarry stool not infrequently accompanies a walled-off perforation, for during active penetration of an ulcer large blood-vessels are not infrequently encountered and corroded. In addition to the history, an ulcerative endocarditis or stasis in the left auricle to account for the source of the embolus should aid in the discrimination. Lead colic is another affection which invites consideration, although the history and findings should render the diagnosis clear.

Nor does this exhaust the list of likely conditions with which protected perforation might be confounded. Extra-abdominal disease, particularly coronary thrombosis with referred pain to the epigastrium, may simulate sealed perforation very closely, indeed. Tabetic crises have not infrequently led to operation for acute gastric or duodenal perforation. Meningitis or pneumonia are not to be overlooked in the list of possibilities, nor hysteria, a case of which came to my attention but recently in which an individual believed to have had an ulcer presented the classical symptoms of an acute perforation.

Treatment.—The therapeutic indications are rendered clear by what has already been stated in connection with the nature of the underlying process. If the symptoms and findings signify that the defect is protected and the process is localized, operation should not be resorted to, but the patient kept under close observation. During this period frequent examinations should be made to detect if present any of the signs of free perforation enumerated in the differential diagnosis. However, unless the diagnosis of walled-off perforation can be unequivocally established and the presence of an acute rupture into the free ab-

dominal cavity ruled out, it is safer to explore than to await further developments.

A large proportion of the patients are seen after the stormy initial symptoms have subsided. Here again, if one cannot safely eliminate the presence of the quiescent period of a ruptured ulcer into the free abdominal cavity, operation should be undertaken at this time. If, however, the presence of a walled-off lesion can be confidently predicted the patient should be placed at absolute rest and rectal alimentation instituted. If at this stage exploration is resorted to and the perforation found to be covered, there is no need, as Lund suggests and Moynihan concedes, to expose and then close the rent afresh. Both these writers recommend in the event that an operation is undertaken a gastroenterostomy, leaving if possible the ulcer and secure barriers untouched. The advisability of surgical manipulation at this time is open to question. Whether or not surgical procedures have been carried out the subsequent treatment consists of instituting accepted methods of medical management for peptic ulcer.

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CLINIC OF DR. L. C. GATEWOOD

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DUODENAL ULCER COMPLICATED BY NEPHRITIS AND ALKALOSIS

THE diagnosis of the typical case of duodenal ulcer with its characteristic history of a dull, steady pain localized to the epigastrium, occurring with a regular time relation to food taking, relieved by food and by alkali, and with its characteristic roentgenologic findings, is now so well understood throughout the medical world as to need no comment, and the treatment by the neutralization of the free acidity, through the writings of Sippy and a host of others, has been so well standardized as to be familiar everywhere. In the case, however, of the patient who does not present the usual symptoms or findings or who presents unusual complications, some very interesting problems in diagnosis and treatment occur, and the cases presented in this group offer a few examples of one of the problems encountered.

Case I.—Mr. F. H. T., age seventy-four, entered the hospital on March 9, 1924, with a history of epigastric pain beginning two years previously and lasting for a period of six or seven weeks, disappearing at the end of that time, but returning some months later, and during the past two years the patient has had four attacks, each time lasting longer and growing more severe. The pain comes on regularly each day three or four hours after food taking. It is absent in the morning before breakfast, has never occurred immediately after eating, is regularly relieved by food taking, and is not associated with desire for bowel movement nor relieved by defecation. He has never tried alkalies for re-

lief. Appetite is good. Diet has been general and he does not know of any foods that make the pain worse. Sleeps well except for nocturia once or twice each night, and for distress of the type described above, which is often severe enough to prevent him from going to sleep during the first two or three hours after going to bed, and occasionally wakens him later in the night. His weight is stationery at 130 pounds. Previous illnesses include typhoid fever at age of twenty-six, sinus infection for a period of several years, relieved by operation at age of thirty-five, and prostatectomy twelve years ago. The patient has been a man of abstemious habits, having never used tobacco or alcohol. He is still in active direction of his manufacturing business, and has been in the habit of walking five to ten miles once each week.

Physical Examination.—Pupils equal and react normally. Teeth have all been removed except four, and these show no evidence of infection. Tonsils are small and free from evidence of infection. Chest of emphysematous type, but with good respiratory excursion and no abnormal dulness or râles. Heart—right border under sternum. Left border 1 cm. outside midclavicular line. Rate and rhythm normal. No murmurs. Aortic second sound accentuated. Liver barely palpable just below costal margin. Spleen not palpable. There is localized tenderness in epigastrium, and increased muscle resistance, but no palpable mass. Stomach was distended with sodium bicarbonate and tartaric acid and the lower border of tympany was 3 cm. above umbilicus. Low-grade peristaltic waves were seen moving from left to right. Right inguinal hernia. Extremities negative for edema and arthropathy. Patellar reflexes normal. Blood-pressure—systolic 156, diastolic 85. Hemoglobin 78 per cent. (Dare). Leukocytes 6700. Urine clear, straw colored, acid reaction 1014. No albumin, sugar, or casts. Ewald test-meal showed HCl 65, total acidity 88; no occult blood. Upon fluoroscopic examination there is a definite deformity of the duodenal cap with outward projection on the greater curvature side of the cap near the base.

The patient was started at once on ulcer management of the Sippy type, using two powders, one of which consisted of 10

grains of calcium carbonate and 30 grains of sodium bicarbonate, while the other consisted of 10 grains each of magnesium oxid and sodium bicarbonate. Each of these powders, therefore, was the equivalent in alkali value of 50 grains of sodium bicarbonate and they were used interchangeably. Upon this dosage the acidity was not controlled, and test aspirations in afternoon and evening showed free acidity varying from 15 to 18, and total acidity from 80 to 110. During the first few days of management the patient was frequently awakened during the early part of the night with distress, and aspiration at such times showed clear watery material with free acidity from 80 to 95 and total acidity 10 to 15 points higher. At no time was a large amount of night secretion obtained, and after a few days of management the night distress disappeared and did not recur. At the outset of management blood chemical determination showed figures approximating the upper limits of normal for the various nitrogen bodies. The first two weeks of management were uneventful, but after that time the patient began to complain of a dull headache which he attributed to his eyes because he had been reading a great deal while in the hospital with glasses which were not properly fitted. However, a blood chemical analysis at this time showed a definite increase in urea and total non-protein nitrogen as shown in Table 1, and the alkali powders were at once stopped.

TABLE 1

BLOOD CHEMISTRY

Date.	Urea.	Uric acid.	Creatinin.	Total N. P. N.	CO_2.	Blood-sugar.	Blood chlorids.
3/24/24	64	2.9	1.75	41	64.5		
3/28/24	55	2.5	1.25	41	60.7		
4/ 3/24	75	4.1	1.50	64	40.9	....	450
4/ 7/24	69	4.0	1.50	52	49.4	390	
4/11/24	78	3.9	1.55	60	45.7	....	470
4/16/24	57	5.4	1.5	43	44.7		
4/21/24	47	4.7	1.45	42	57.0	....	480
4/26/24	42	4.3	1.4	38	56.0		
4/30/24	43	3.6	1.4	37	58.9	....	490
5/ 5/24	40	3.8	1.4	38	63.6		

After a rest period of two days he was started on powders containing 15 grains each of tribasic magnesium phosphate and calcium phosphate, as suggested by Shattuck.[1] Five days later blood-urea had increased from 55 to 75, and alkali was stopped entirely. The patient continued with hourly feedings of milk and cream and no other effort was made to control his acidity for a period of two weeks, during which time he remained practically free from distress, but control aspirations of stomach content continually showed free acid from 70 to 100 and total acid 90 to 130. In the meantime blood nitrogen had gradually decreased until blood urea was 42, uric acid 4.3, creatinin 1.4, non-protein nitrogen 38, CO_2 56, and chlorids 480. After a few days his diet was enlarged to include three small meals of simple food with milk and cream, alternating with powders between meals, and the patient continued to follow out the usual diet régime of ulcer management in this way with alkali powders containing only 10 grains of sodium bicarbonate and with a rest period of five days at the end of each five or six weeks. On this régime blood urea has remained consistently between 35 and 45 mgm. per 100 c.c. of blood and other nitrogen bodies at corresponding levels. Whenever urea has increased above 40 mgm. the alkali has been discontinued for a few days and then resumed on the previous schedule. Plasma CO_2 has varied from 55 to 47 regularly as compared with normal figures of 53 to 65, showing a tendency to decrease when nitrogen retention has increased. This is a characteristic finding with nitrogen retention due to a nephritic process, there being, as a rule, a low-grade acidosis indicated by a decreasing plasma CO_2, associated with an increase in nitrogenous waste in the blood. Blood chlorids, as a rule, remain normal.

Upon this régime the patient remained free from ulcer symptoms and had practically none of the headaches experienced during his stay in hospital when nitrogen retention was more marked. He has been able to continue his business without interruption, and upon gradually discontinuing this type of management at the end of ten to twelve months he remained free from symptoms referable to the gastro-intestinal tract. He has,

however, continued to have occasional headaches. Blood urea has consistently remained between 35 and 45, but, as a rule, near the lower figure, and its increase to higher levels has only followed more strenuous activity or intercurrent infection, such as cold taking, and has been, as a rule, associated with low-grade headaches. Throughout this entire period of observation urine has been examined regularly and no albumin has ever been found. Specific gravity has varied, as a rule, between 1010 and 1016, although there have been times when the patient was taking alkali when the specific gravity reached 1020 and, on one occasion, 1030. In only a very few of the many specimens examined have casts been found and then only very few. The patient presents himself for examination at the end of a period of three years' observation with the following findings: Blood-pressure, 136/80; blood urea, 49; uric acid, 3.6; non-protein nitrogen, 37. This is the highest nitrogen level discovered at any time since his stay in hospital. Urine is now free from albumin and casts.

The problem in this case was that of treating an active ulcer in a patient who was unwilling to undergo operation and to whom we could not urge it because of his age and the risk involved. The picture was that of an individual whose capacity for kidney output had decreased with advancing years until it was barely equal to the load imposed upon it by normal nitrogen metabolism, and in whom the excretion of a considerable amount of the salts produced by the combination of alkali with acid in the stomach was sufficient to precipitate a more acute degree of nitrogen retention with attendant clinical symptoms. He had had no previous evidence of kidney insufficiency, presented no urinary findings, and no abnormal retention of nitrogen waste on admission. There was, therefore, no apparent contraindication to the usual method of ulcer treatment. This case serves to illustrate the necessity for being continually upon the alert to recognize symptoms of renal insufficiency in patients upon ulcer treatment and to check all such findings by means of blood-nitrogen determinations. By careful watching it has been possible not to control this patient's acid in the usual sense, but to keep it within such range as had made possible the healing of his ulcer, so that he

has now been free from symptoms during a period of two years in which he has had no alkali and has taken a general diet.

Case II.—Mr. P. P. C., age sixty, first presented himself for examination October 21, 1921, complaining at that time of epigastric distress which had recurred for periods of a few days to several weeks in duration since aged seventeen. During the preceding five years this distress had been present probably one-fourth of the time. He gave a typical history of dull, steady epigastric distress coming on regularly from one to three hours after eating, never occurring in the morning before breakfast nor immediately after food taking, and never continuing later than six hours after a meal. It was regularly relieved by food taking and by alkali, and not associated with cramp-like pains in the abdomen or desire for bowel movement, and not affected by bowel movement. He described also dizziness associated with tinnitus and deafness in the left ear for a number of years, and gave a history of prostatic enlargement diagnosed and treated by a competent urologist who did not regard the condition as sufficient to justify prostatectomy.

Physical Examination.—The heart was moderately enlarged with the left border 12 cm. from midsternal line, and right border just beyond the sternal margin. There were numerous extrasystoles, but no murmurs were found. The lungs were normal. Liver was not enlarged and there were no signs of heart decompensation. Prostate moderately enlarged, but no nodules palpable. Blood-pressure, systolic 150, diastolic 96. Ewald test-meal showed free HCl 30, total acidity 100, fresh blood produced by traumatism at the time of aspiration accounting for the large proportion of combined acid. Urine was negative for albumin, sugar, and casts. Fluoroscopic examination at that time revealed no abnormality of stomach or duodenum, and the patient was discharged upon a moderately restricted diet and directed to report at once if he had a recurrence of his distress.

The patient continued with essentially the same symptoms until March 23, 1923, when he was admitted to the hospital with a history of having vomited about a quart of dark, partly clotted

blood two hours previously. He complained of weakness, thirst, and dizziness, and was quite pale. Blood-count at this time showed hemoglobin 56 per cent., leukocytes 15,300. Blood-pressure, systolic 102, diastolic 56. Urine clear, acid reaction, specific gravity 1012; albumin, sugar, and casts absent.

The patient was put to bed with an ice coil over the abdomen and was given a hypodermic of morphin. Nothing was given by mouth except calcium carbonate and sodium bicarbonate (60 grains each) every hour during the first three hours and thereafter every two hours, continuing throughout the night and the following day, for the purpose of neutralizing the acid secreted in response to the stimulus of blood in the stomach content. No further vomiting having occurred and the patient being free from nausea on the second day, feeding was started with 1 ounce of milk and cream each hour from 7 A. M. to 7 P. M., and with alkali midway between feedings. On this régime the patient complained slightly of nausea, but had no further vomiting. However, he continued to have tarry stools. Because of the previous history characteristic of a chemical type of distress and the probability of persistent night secretion, the alkalies were continued throughout the night during the early days of management, and the patient remained free from the old type of abdominal distress. However, he complained of headaches and slight nausea, and blood chemical determination on April 3d showed urea 66, non-protein nitrogen 53, creatinin 2.5. Fearful to discontinue the alkali because of the patient's persistent bleeding, the same régime was continued through the four succeeding days and blood chemical determination at that time showed urea had increased to 105 mgm. and creatinin to 3.2, as indicated in Table 2. On April 6th stomach fluoroscopy revealed a narrowed and incompletely filled area in the terminal 2 or 3 inches of the antrum of the stomach. Re-examination on April 10th gave identical findings, raising, therefore, a definite question of the possibility of carcinoma of the stomach as the cause of the continued bleeding. Meanwhile the patient had become somewhat disoriented and at times presented hallucinations, and on April 11th another blood chemistry showed urea

TABLE 2

BLOOD CHEMISTRY

Date.	Urea.	Uric acid.	Creatinin.	Total N. P. N.	Blood-sugar.	Carbon dioxid.
3/27/23	....	....	....	....	....	60.5
4/ 3/23	66	5.2	2.5	53	112.5	27 (whole blood)
4/ 7/23	105	7.4	3.2	76	142	
4/11/23	180	11.4	4.3	118	220	
4/14/23	105	5.0	3.2	70	170	
4/17/23	54	3.3	1.75	36	130	
4/26/23	26	3.3	1.35	25	147	
5/ 4/23	26	3.5	1.40	28	135	

180, uric acid 11.4, creatinin 4.3, and non-protein nitrogen 118. Urine at this time showed only a trace of albumin and a few casts, but the clinical symptoms together with the blood chemical findings were unquestionably those of uremia and all alkalies were immediately discontinued. In the succeeding six days blood-nitrogen gradually decreased until on April 17th blood-urea was 54, non-protein nitrogen 36, and creatinin 1.75. During the entire time the patient continued to pass stools which were strongly positive for blood, many of the stools being tarry in appearance. Because of the continued bleeding and the *x*-ray findings so suggestive of carcinoma of the pyloric antrum, on April 18th an exploratory laparotomy was performed by Dr. A. D. Bevan under nitrous oxid anesthesia, and a very thorough examination of stomach and of all of the abdominal viscera failed to reveal any evidence of carcinoma or ulcer of the stomach or of any other recognizable pathology. The patient made an uneventful recovery from his laparatomy, and by the tenth day after operation his blood-nitrogen retention had still further decreased until blood-urea was 26, non-protein nitrogen 25, and creatinin 1.35. He remained in hospital until May 5th, and was discharged at that time with stools still giving a positive benzidin reaction for blood, although no longer dark in color.

He returned to hospital for observation a year later, and fluoroscopic examination again gave identical findings with in-

ability to fill the terminal portion of the antrum. Blood chemical determination at this time showed urea 37, uric acid 5.1, creatinin 1.35, non-protein nitrogen 34, CO_2 51.3, blood-sugar 145. After leaving the hospital the patient was placed upon 10-grain doses of sodium bicarbonate two or three times after each meal and on this régime remained free from his old type of distress. When, however, he had discontinued the alkali for a few days, he would frequently have a return of his old type of abdominal pain. This gradually disappeared and he has remained entirely free from any abdominal distress. His stools have remained free from blood. Blood-pressure has remained consistently about 150 to 160 systolic, and 90 diastolic, and blood chemical determinations have shown nitrogen figures just above the limits of normal. Urine has at times shown a trace of albumin and an occasional hyaline and granular cast, and has at other times been free from both albumin and casts.

This patient is quite similar to Case I, with a moderate increase in blood-pressure and evidence of cardiac hypertrophy. He had been entirely free from evidence of renal insufficiency, and the only previous urinary symptoms had been referable to prostatic enlargment of moderate degree. There had been no previous evidence of nitrogen retention. However, on large doses of alkali in attempting to control acidity as a means toward arresting hemorrhage, he promptly developed nitrogen retention which progressed to a high level (blood urea 180), at which time the patient showed disorientation and other uremic manifestations. Blood-nitrogen returned approximately to normal soon after discontinuing the alkali, and it was not subsequently deemed safe to give him doses of alkali sufficient to control his acidity, although he continued to have an acid type of distress at intervals, and stomach analyses showed a high acidity.

Case III.—Mr. R. T. L., age twenty-nine, entered the hospital January 12th because of epigastric pain which was of two types, the one dull and aching in character, rather sharply localized and occurring with the usual time relations of ulcer distress, and the other more diffuse and cramping in character and diffi-

cult for the patient to localize. The dull, gnawing pain had been present most of the time for seven or eight years, though relieved temporarily by various types of management and always relieved by alkali or by food taking. The patient stated, however, that he had never been able to follow consistently any type of ulcer management because whenever he had taken alkalies regularly he had begun to have persistent nausea and vomiting and had to discontinue their use. In addition to the above complaints the patient gave a history of osteomyelitis beginning twelve years ago, and he has been free from discharging sinuses only for short periods during the past several years. He has at the present time a point of pain and tenderness over one femur and states that from previous experience he believes an old healed sinus is about to reopen at this point.

Physical Examination.—Patient is moderately emaciated, and shows many scars of previous operations and draining sinuses, particularly in the region of the left hip. The skin is soft, smooth, and moist. Head, face, eyes, ears, nose, and throat are normal. Thorax is well developed with good respiratory excursion, and no abnormal areas of dulness, tubular breathing, or râles. The heart borders are normal, rhythm regular, and tones clear. Liver, spleen, and left kidney are not palpable. Right kidney palpable. There is moderate tenderness in the epigastrium. On palpation over the epigastrium the stomach assumes form and low-grade peristaltic waves are seen moving across the epigastrium from left to right. Extremities show the scars of operations and discharging sinuses from old osteomyelitis, and an area of tenderness and redness over the left femur in the neighborhood of an old healed sinus. There was no edema of feet or ankles and reflexes were normal. Blood-pressure, systolic 110, diastolic 76. Hemoglobin 79 per cent. Erythrocytes 3,980,000, leukocytes 6900. Urine acid, sugar 0, albumin trace. Microscopic examination showed hyaline and granular casts, pus, and epithelial cells. Phenolsulphonephthalein injected intravenously for a kidney function test gave a secretion of 65 per cent. first hour; 15 per cent. second hour; 3 per cent. third hour; total 83 per cent. Blood chemistry, urea, 113,84;

uric acid, 7.24; creatinin, 3.42; non-protein nitrogen, 97.56; CO_2, 98; chlorids, 300. Within a few hours after entering the hospital the patient vomited considerable quantities of watery greenish material containing no free hydrochloric acid. He presented thus the clinical picture of alkalosis, the characteristic symptoms of this condition being nausea with vomiting of large amounts of watery material containing, as a rule, little or no hydrochloric acid, and the characteristic blood chemical findings including nitrogen retention, increase in alkali reserve, and marked lowering of blood-chlorids. The term "alkalosis" has given rise to an erroneous impression in the minds of many that it is dependent upon the administrations of alkali, and while it is true that when this condition is present it is markedly aggravated by the administration of soluble alkali, as we see in this case it may develop spontaneously in an individual with obstruction who has received no alkali.[2, 3, 4]

The following day the patient was fluoroscoped, showing a very large stomach with markedly active peristalsis closing off completely to the end of the antrum, and with so little barium passing through the pylorus that it was impossible to visualize the duodenal cap.

Because of the high-grade alkalosis it was impossible to make any further examination of the patient at this time, and he was started immediately on retention enemas of salt solution containing 4 per cent. glucose and was given normal salt solution by hypodermoclysis. The following table 3 will show the course of the process as demonstrated by blood chemical determinations, showing a rapid decrease, so that within six days the blood urea was down to 31.26 and chlorids to the normal level (487.5). It is interesting to note in this determination (1/21/26) that alkali reserve has dropped below the normal level to 44.7, and through the next two weeks remained continually below normal while blood-nitrogen remained continually above normal and chlorids were normal or above. This, then, is the characteristic picture of retention of nitrogen products due to renal damage as distinguished from that retention which occurs on the basis of alkalosis and which is associated with an increase in

TABLE 3

BLOOD CHEMISTRY

Date.	Urea.	Uric acid.	Creatinin.	Total N. P. N.	CO_2.	Blood-sugar.	Blood chlorids.
1/15/25	113.84	7.24	3.42	97.56	98.0	....	300
1/16/26	71.62	5.19	1.87	59.68	81.8	....	360
1/18/26	71.04	4.70	1.81	57.56	72.1	....	420
1/21/26	31.26	2.56	1.33	32.00	44.7	....	487.5
1/26/26	38.36	3.04	1.32	35.76	44.3	....	480
2/ 5/26	33.13	3.64	1.39	32.24	43.30	....	610
2/ 8/26	....	....	....	....	72.9		
2/ 9/26	18.18	4.24	....	38.84	67.3	....	440
2/13/25	26.72	3.80	1.48	44.44	44.3	....	452
2/19/26	15.8	2.68	1.20	32.52	43.8	....	515
3/ 1/26	14.38	2.88	1.20	30.28	46.2	....	490
3/ 5/26	19.90	3.32	1.45	48.56	53.2	....	455
3/ 8/26	16.66	3.12	1.26	34.48	44.7	76.9	465
3/14/26	12.12	2.96	1.22	34.32	43.8	89.3	465
3/20/26	10.68	2.48	1.23	32.64	57.0	93.4	445
4/ 7/26	13.16	2.67	1.28	30.64	41.9	....	485
4/14/26	15.50	2.83	1.25	33.04	51.0	89.7	425

blood CO_2 and a decrease in chlorid. The patient gave a history of having had albumin and casts in the urine during the preceding two or three years, and with a history of long-continued pyogenic infection we had reason to believe that renal damage had resulted from this infection producing a moderate degree of renal insufficiency. At the end of two weeks the patient had so far improved that we were again able to make a fluoroscopic examination, and at this time barium passed readily through the pylorus and we were able to demonstrate a definite deformity of the duodenal cap with an out projecting shadow from the greater curvature side of the cap extending medially toward the antrum of the stomach, giving the characteristic picture of a pouch such as is formed by ulcer perforation. The problem then was the management of a chronic recurrent ulcer with perforation and a moderate degree of pyloric obstruction in a patient who, as a result of long-continued pyogenic infection, had suffered sufficient renal damage to render him intolerant to alkali. Since he had had a

marked alkalosis on admission to the hospital without previous alkali treatment, we might reasonably expect that the administration of alkali in an attempt to control acidity would precipitate a more dangerous degree of alkalosis. An attempt was made for a few days to carry on ulcer management with the tribasic magnesium and calcium phosphate suggested by Shattuck, but the patient promptly developed the previous symptoms of alkalosis and a corresponding change in blood chemical findings, and this medication had therefore to be abandoned. The physical condition of the patient was such that he could not be regarded as a good surgical risk. On the other hand, it was possible to maintain water balance and normal blood acid alkali balance only by the continual administration of salt solution by rectum or subcutaneously, and it was manifest that the patient could not indefinitely continue on this basis. Operation was, therefore, advised as the lesser risk, with the full realization that it often precipitates a more marked degree of alkalosis than that previously experienced. On March 3d, under ethylene anesthesia, the abdomen was opened by Dr. Gatewood. The lesser peritoneal cavity was found obliterated by adhesions and the first portion of the duodenum and pyloric end of the stomach were adherent to the liver and gall-bladder. The duodenum was indurated about the site of a chronic ulcer. The pyloric end of the stomach was amputated, the proximal end of the duodenum invaginated, and a modified Polya operation was done, so that the ulcer in the duodenum was covered in and was left in a blind pouch with no direct connection with the stomach. The patient's postoperative course was stormy, but not more so than had been anticipated. The stomach filled up repeatedly with watery material similar to that previously vomited during the patient's attacks of alkalosis, and if the patient was not aspirated when he complained of fulness he would vomit large amounts of this watery material. Because he had become accustomed to the stomach-tube it was more comfortable for him to be aspirated than to allow material to accumulate to the point of inducing vomiting, and Table 4 shows more clearly than words the course of his water balance during the days succeeding operation. With

TABLE 4

WATER BALANCE

Date.	Intake.				Output.				Excess.	
	By mouth.	By rectum.	Hypodermoclysis.	Total intake.	Stomach contents.	Stools.	Urine.	Total output.	Intake.	Output.
3/ 5/26	210	2160	....	2370	520	...	430	950	1420	
3/ 6/26	....	2500	1500	4000	2050	...	600	2650	1350	
3/ 7/26	690	3000	1200	4890	....	...	550	550	4340	
3/ 8/26	750	2400	1200	4350	3250	...	600	3850	500	
3/ 9/26	240	2880	1200	4320	1850	...	680	2555	1765	
3/10/26	720	2880	1200	5400	2800	800	500	4100	1300	
3/11/26	1650	2400	1500	5550	2200	350	775	3320	2230	
3/12/26	2040	2400	1500	5940	2300	1930	700	4930	1010	
3/13/26	1750	2400	1600	5700	3900	575	700	5175	625	
3/14/26	1760	2400	1540	5700	2900	50	750	3700	2000	
3/15/26	1540	2460	1520	5520	3950	...	760	4710	810	
3/16/26	1510	2880	1500	5890	4910	500	630	6030	...	140
3/17/26	1346	2400	1500	5246	2900	...	600	3500	1746	
3/18/26	1940	2940	1500	6380	3050	...	420	3470	2910	
3/19/26	1520	2920	....	4440	3180	480	530	4190	250	
3/20/26	1980	2480	....	4460	2350	450	930	3730	730	

blood chemical determinations as a guide, and continuing to supply liquid by rectum and hypodermoclysis in amount sufficient to maintain water balance, and with salt in quantity sufficient to maintain normal blood chlorid, the patient's chemical processes gradually re-established themselves upon a normal basis. The excessive secretion of watery material in the stomach gradually subsided and the patient was able to leave the hospital on April 11th free from abdominal distress and with only the occasional accumulation of material in the stomach in quantities sufficient to induce nausea or require aspiration for relief. He was able to take a normal diet without recurrence of his previous distress, and blood chemical determination on discharge showed all nitrogen bodies within the limits of normal.

He returns to the hospital at the present time for re-examination at the end of a period of seven months, during which time he has gained strength and weight so that within the past few weeks he has taken a motor trip of 500 miles. Physical examination is now negative except for the scars of his old osteomyelitis and one small discharging sinus in the left thigh. Blood-examination shows hemoglobin 82 and leukocytes 11,500. Urine is acid in reaction; sugar absent, but albumin present, and occasional

hyaline and granular casts. Ewald's test-meal shows free HCl 20, total acidity 31. Motor meal passed in normal time. Phenolsulphonephthalein excretion was 52 per cent. in the first hour and a total of 80 per cent. in three hours. Blood chemistry showed urea, 42.4; uric acid, 6.1; creatinin, 1.14; non-protein nitrogen, 36.36; CO_2, 55.1; chlorids, 445. The patient presents at the present time, therefore, the findings of nitrogen retention with albumin and casts in the urine as evidence of a chronic renal damage from long-standing pyogenic infection, but is entirely free from gastro-intestinal symptoms and is able to do some office work. His gastro-intestinal emergency has been met, and his physical condition at the present time is as good as may be expected with a chronic recurring osteomyelitis and associated renal pathology.

The 3 patients in this group present a problem very frequently encountered. We have long known that the patient with a well-marked nephritis will not tolerate doses of alkali such as are commonly required to control the acidity of an ulcer patient. It has not been so generally recognized, however, that many patients with only a moderate degree of kidney damage are unable to excrete the necessary quantity of nitrogen waste per day in addition to the quantity of the salts of sodium and calcium produced by the neutralization of hydrochloric acid with calcium carbonate and sodium bicarbonate in the alkali treatment of ulcer. Under such conditions the kidney apparently eliminates the inorganic salts at the expense of developing a nitrogen retention, and it must be recognized that many individuals who have given no previous evidence of renal insufficiency will develop a nitrogen retention of this type on ulcer management. In some cases it is possible by using the tribasic magnesium phosphate and tribasic calcium phosphate instead of the usual forms of alkali, to carry on the treatment without producing a disturbing degree of nitrogen retention. But more often we find that the individual who develops retention on the one type of alkali will do so also on the other. The characteristic symptoms are those of any renal insufficiency, beginning usually with headache and progressing to nausea and vomiting, and the presence of albumin and casts in the urine. If the treatment be con-

tinued, it is entirely possible to produce a degree of nitrogen retention sufficient to cause disorientation (Case II) and other definite symptoms of uremia. The condition being dependent upon a diminished capacity for renal function is, therefore, permanent, and such patients cannot tolerate the usual doses of alkali. Unfortunately, they are also, as a rule, poor surgical risks, and the problem presented in their management is one requiring careful study and nicety of judgment to determine the course of minimum danger to the patient. In 2 of these cases it has been that of a milk and cream diet with very small doses of alkali sufficient only to lower the acidity and not to control it. In the third case the addition of alkalosis to the picture by reason of the patient's high-grade obstruction made operation, hazardous as it might seem, the safer course to follow.

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ESTIMATION OF THE SEVERITY OF THE DIABETIC DEFECT—TWO CASES

IN the practical management of diabetes it is important to have some easily applied standards for estimating the severity of the defect. If at the first conference with the patient one can approximate this, valuable time will be saved, and errors which may be costly will be avoided. In searching for such standards it is, of course, recognized that the estimates will be only approximate ones, since it requires from ten days to two weeks to measure with any accuracy a patient's tolerance. However, any management that is adopted must make some assumptions, and it is with the basis of such assumptions that we are concerned. How then shall we proceed in any given case? Is the case sufficiently severe to demand the use of insulin? Can it be handled by diet adjustment alone? What type of diet shall be used; one with a high ratio and therefore a high fat diet; or one with a low ratio and consequently a diet relatively rich in glucose? Experience has shown that the average medical student who has completed a clinical course in diabetes is quite confused when presented with practical considerations of this type. It is the purpose therefore of the present clinic to present 2 cases which will illustrate the principles involved. One of these patients was desugarized easily by diet adjustment, but the other required the use of insulin and a diet of an entirely different type. In each case criteria are presented for the determination of the severity of the diabetic defect.

Case I.—The patient, a male Hebrew, age thirty-six, peddler by occupation, presents the following history:

Present Complaint.—1. Numbness of the hands and feet associated with sensations of prickling with pins and needles. Left leg feels dead.

2. Polydipsia, polyuria, and polyphagia.

3. Loss of 55 pounds in weight.

4. Hoarseness and cough.

Onset and Course.—The patient was well and healthy until one and a half years ago. At this time he noticed the paresthesias described above. These have persisted since onset and gradually grown worse in last six months.

The polydipsia, polyuria, polyphagia, and the loss of weight were first noticed six months ago, but have become acute in the last month. He now complains bitterly of the dry tongue.

The hoarseness and cough are attributed by the patient to his occupation (peddler) and to his exposure to the winter weather. He finds that with rest his voice improves and that the cough is better in the summer.

Within the last three weeks there have been recurring attacks of epigastric pain, associated with nausea, occasional attacks of vomiting, flatulence, and constipation.

The history otherwise is not important.

Physical Examination.—The patient is obese, appears quite comfortable, and is not acutely ill. The essential points in the examination are:

Size: Weight, 196¼ pounds; height; 5 feet, 3¼ inches, surface area, 1.98 square meters.

Skin: Dry, scaly. Over the elbows and knees are well-developed lesions of xanthoma diabeticorum. Bluish discoloration to toes.

Tongue dry.

Larynx: Chronic laryngitis.

Blood-pressure 134/74.

Examination otherwise negative.

Laboratory Findings.—Urine: Specific gravity 1039.

Albumin: +.

Sugar: Quantitative estimation based on patient's story of output shows that he is passing 125 to 150 grams of glucose per day.

Acetone 0.

Diacetic 0.

Hyaline and granular casts present.

Blood: Hemoglobin, 91; red blood-cells, 4,400,000; white blood-cells, 10,500; sugar, 228; N.P.N., 38; carbon dioxid, 56.

Discussion.—Here is a relatively young man (age thirty-six) who shows all the evidence of a recently acquired diabetes. He is in a stage of rather acute water disbalance. The dehydration is shown by his history, by the dryness of the tongue, and by the slight concentration of blood (hemoglobin 91 per cent.). The loss of 55 pounds in weight over a period of six months is certainly quite striking even in an obese patient. Consequently one would say that we are dealing with a rather acute and doubtless severe case of diabetes. The obesity alone speaks against this conclusion, for we have been taught that diabetes runs a milder course in the obese. It should still be remembered that many of our severe diabetics tell us that once they too were fat. The really important diagnostic feature in the case is the urine examination. This shows a high specific gravity (1039) and the quantitative estimation (by titration which can be done in a few minutes) indicates an output of 125 to 150 grams of sugar in twenty-four hours. In other words, the urine is abundant and practically saturated with sugar. It is next noted that albumin and hyaline and granular casts are present. This may simply mean that the kidney is suffering from dehydration as the remainder of the tissues are, or it may indicate a more serious defect. The significance, therefore, of the casts and albumin cannot at present be determined. However, the absence of acetone bodies at once reassures us, and the assumption is made that the case is not so severe as it appears.

The significance of acetone bodies in the urine of a patient on an uncontrolled diet, usually a high carbohydrate one, can best be appreciated from the following discussion. Hill and his associates maintain that the ultimate source of energy for con-

traction of the voluntary muscle is carbohydrate. When the work is light there is sufficient carbohydrates stored in the muscle to furnish this energy and the respiratory quotient is 1. With more prolonged work the respiratory quotient falls, showing the utilization of the fatty acids and protein. However, he maintains that the fatty acids and the protein are used for muscular work only after conversion into glucose or a carbohydrate body. Whether carbohydrate bears this same fundamental and indispensable relationship to the vegetative activities (secretion absorption, excretion, etc.) has not been determined. It is safe to say that while it may not play an indispensable rôle, it certainly plays an important one. In spite of the large amount of carefully controlled experimental data which has been presented in support of this view, and in spite of its general acceptance, Lusk and his associates have not been willing to adopt it. Lusk, however, does not deny the fundamental rôle of carbohydrate as a source of energy for muscle contraction. He is simply unwilling to concede that fatty acids must first be converted into carbohydrate moieties before oxidation. According to either view there is a minimal quantity of carbohydrate which must be oxidized. The size of this quantity would vary according to the view adopted. In practical terms these views explain to us why life is not possible in the presence of complete loss of oxidative capacity for carbohydrates. We can further understand that the minimal amount of carbohydrate, which must be burned, bears a very direct relationship to the size of the body and the amount of voluntary muscular activity. We may then say that there is for a given body, of a given size, age, and sex, and doing a given amount of work an *indispensable minimum carbohydrate* or *glucose requirement*. The fact that a patient is a diabetic does not relieve him of the necessity of burning this indispensable carbohydrate moiety if he would live. When he is able to oxidize such a quantity of glucose, he then has the power to derive energy from the oxidation of a quantity of fatty acids three times the weight of the glucose oxidized. As a further corollary to this view it must be appreciated that the fatty acids are not commandeered for energy until a deficiency of glucose exists at

the site of the work. When acetone appears in a diabetic on an uncontrolled diet, it means that the energy required of the body is greater than can be supplied by the oxidizing glucose and a quantity of fatty acids three times the weight of the glucose. These extra fatty acids in their oxidation from a sixteen or eighteen carbon chain down to the four carbon stage yield the necessary extra energy to maintain it, but they leave behind the toxic acetone bodies. Consequently their appearance is a red flare signal which the body is waving to us. There should be no mistaking it. Under the existing energy demands that particular body is already a metabolic bankrupt. In the absence of acetone bodies we may realize that any or all of a patient's symptoms may be important or even serious, but they need not alarm us. If he is not an arteriosclerotic diabetic, he can be desugarized by diet methods. What then is the difficulty with our patient? This man's appetite has led him to eat carbohydrate in excess of his tolerance, and the body tissues and fluids are loaded with this excess of glucose which acts as a foreign body. The fluid balance has been disturbed by this foreign crystalloid (glucose) and his symptoms are arising from this disturbance. This same condition could be induced in a normal person by the intravenous injection of glucose in excess of the normal tolerance. The problem, therefore, is to reduce the intake of glucose to the level of his tolerance.

Subsequent Course of the Case.—On entrance to the hospital the patient was placed on a basal plus 10 per cent. diet, and confined to bed. The caloric requirement of this diet was 1986 calories. If we assume that 10 per cent. of the calories of such a diet is to be derived from protein, and if we assume that the body will burn the theoretical 3 grams of fatty acids to every gram of oxidizing glucose, then the minimal glucose value of this diet which will prevent a significant acidosis must be at least 63 grams. In practice it is quite difficult to use diets with such a high ratio, although they may be theoretically satisfactory. Consequently a diet (CHO. 50; P. 55; F. 174) which furnished a glucose value of 100 with a ratio of fatty acids to glucose of 1.8 was ordered. Within twenty-four hours the patient desugarized,

and his fasting blood-sugar gradually fell to a level of 105 mgm. per 100 c.c. of blood. He left the hospital on a diet containing CHO 100; P. 96; F. 254; calories 3070; glucose 181; fatty acids 282; ratio 1.5. This diet represents a basal plus 70 per cent. He has remained sugar free for a period of two months since discharge, and this diet has proved adequate for his work. This case illustrates the method of desugarization by means of a diet with a relatively high ratio of fatty acids to glucose.

Case II.—The second patient, a white female, age fourteen, gave the following history:

Present Complaints.—Polyuria, polydipsia.
Extreme weakness.
Loss of weight 24 pounds in one year.
Headaches.
Abdominal cramps.
Palpitation of heart.
Dyspnea on exertion.

Onset and Course.—The patient was a healthy school girl one year ago (December, 1925) weighing approximately 90 to 95 pounds. About this time she developed polyuria, polydipsia, headache, and abdominal cramps. She consulted a physician, who made a diagnosis of pulmonary tuberculosis. About a month later she was examined at one of the dispensaries of the Municipal Tuberculosis Sanitorium. Here the diagnosis of tuberculosis was confirmed. However, the patient states that there was no examination of urine. In March, 1926 she had grown so weak that she was confined to bed and was unable to make the regular trip to the dispensary for observation. She remained a bed patient from March until July, 1926, and at this time her strength had recovered sufficiently for her to be out of bed and take short walks on the outside. There has been little change in weight since March. The polyuria and polydipsia have diminished somewhat. However, her strength has not returned and she tires on the least exertion. In October, 1926 the Health Department insisted on hospitalization because there were other small children in the home. She entered the dispensary of the

Research Hospital, where a diagnosis of diabetes mellitus was made and she was at once transferred to the hospital. The system history was essentially negative.

Physical Examination.—The patient, an emaciated white female, sits quietly, exerts herself as little as possible, and answers questions slowly.

She weighs 71 pounds and is 60 inches tall. The normal weight of a girl of this height and age is 98 pounds. The skin and tongue are quite dry. Scapulæ winged. Respirations 24. The apex-beat is quite diffuse, left border of heart displaced outward 1½ fingers, rate 120, rhythm regular, soft systolic murmur at apex. The liver is palpable 3 to 4 fingers below costal margin. Patellar reflexes elicited with difficulty. The examination otherwise was negative.

Laboratory Examination.—Blood counts: Hemoglobin, 98; red blood-cells, 5,050,000; white blood-cells, 9450.

Differential normal.

Blood chemistry:

Sugar, 286; N.P.N., 32; carbon dioxid, 30.

Urine:

Specific gravity 1035.

Sugar present (5 c.c. Hain's solution ppt. with 1 gtt.).

Acetone +++.

Diacetic ++.

Albumin 0.

Casts 0.

Discussion.—The history of this case is extremely interesting because it shows how this girl's life was saved by weakness. When she could not go to the dispensary she stayed at home. This lessened her needs for energy and reduced the *indispensable minimal glucose requirement* to a lower level. When she could not live longer at an ambulatory level she went to bed, and from necessity adopted a therapeutic measure which preserved her life.

Holt has estimated that for girls of this age and size the caloric requirements per kilo are approximately 74 calories. Of this, 34 calories per kilo represents basal requirements, 7 growth,

26 activity' and 7 excretion. To some this allowance may seem high, but it emphasizes the enormous saving to the young diabetic of curtailing his activities. If we assume that this patient weighed in March 80 pounds (36 Kg.), then, using Holt's standards, we may say that her caloric requirements were reduced from 2664 to 1728 calories by bed rest. If now we figure the minimal theoretical glucose necessary to support this metabolism, using the test-tube ratio of 3, we find that this minimal glucose value was reduced from 94 grams per day to 61 grams per day. This certainly is quite a saving in the case of a severe diabetic.

We may conclude that from the months of March to October her metabolism had adjusted itself to the lowest possible level, and that even at this level she was not quite able to burn sufficient glucose to catalize the metabolism completely. Additional quantities of fatty acids were being burned with the gradual accumulation of acetone bodies, depletion of the alkali reserve, and the production of chronic acid intoxication. All of this information was obtained from the patient's history and from the urine examination. The examination of the blood after entrance to the hospital simply defined the degree of the acidosis and confirmed the concentration of the blood (hemoglobin 98, red blood-cells 5,050,000).

Subsequent Course.—It is perfectly obvious that this girl requires food and the energy which it furnishes.

She is, therefore, an insulin case. It is also evident that at present she requires above all things the oxidation of a liberal quantity of glucose. Experience has shown that a child should not be fed a diet with a ratio above 1 or 1.2 until it is demonstrated at a subsequent time that that particular child is able to oxidize fat in larger quantities.

This girl's diet was planned on the assumption that she weighed 42 kilos (92 pounds), and on a basis of 48 calories per kilo. On Holt's scale this would furnish sufficient energy to cover all requirements except those of activity. The specific order was CHO. 90; P. 50; F. 160; calories 2000; G. 135; FA. 167; R. 1.2. Insulin was given at first twice daily and later three times daily until patient was desugarized. She remained in the hospital for

two months. The diagnosis of tuberculosis was disproved by the course of the case, by the physical findings, and by repeated sputum examinations and *x*-ray plates. Her weight increased from 71 to 95 pounds. She was discharged from the hospital with a normal blood-sugar, on 40 units of insulin in the morning and 25 units in the afternoon. The diet was the same as the admitting one.

This case illustrates the use of insulin and a relatively high glucose diet with a corresponding low ratio (1.2).

CLINIC OF DR. GEORGE O. SOLEM

LUTHERAN DEACONESS HOME AND HOSPITAL

MASSIVE COLLAPSE OF THE LUNG

MASSIVE collapse of the lung is defined by Bradford[1] as "an unusual condition in which the lung without the presence of any gross lesion, such as bronchial obstruction, pleural effusion, etc., interfering with the free entry of air, becomes airless to a greater or less degree and therefore useless for respiratory purposes. This collapse involves varying amounts of the lung in different cases, usually an entire lobe or a very large portion of one lobe and not infrequently the whole of one lung."

The recent literature has had reports of numerous cases usually following abdominal operations or chest injuries and the statement having been made that massive collapse has not followed injuries of the head and neck[1] or tonsillectomy,[2] we were stimulated to report the following case:

Miss F. R., age twenty-two, was seen at the Lutheran Deaconess Home and Hospital in consultation with Dr. R. M. Hutchison, to whom we are indebted for this report. On September 23, 1926 the tonsils were removed under general anesthesia at another hospital, and that evening she complained of pain in the left side and cough. She was allowed to go home the next day and remained in bed for three days before Dr. Hutchison was called. The pain and cough decreased, but the patient continued to have some fever and dyspnea. She entered this hospital October 2d, at which time we were asked to see her. The temperature was 99.8° F., pulse 90, respirations 28. She was coughing considerably, but there was no expectoration. There was some pain in the left chest.

The patient did not appear acutely ill, but there was slight cyanosis and some dyspnea, especially on changing position.

The left chest seemed retracted and there was a definite limitation of expansion. Percussion revealed a dullness over the entire left side of the chest except just to the left of the spine below the spine of the scapula, where there was slight resonance. Dullness was most marked at the base posteriorly. The apex-beat was seen and felt in the midaxillary line in the fourth interspace. The right heart border was just to the left of the left sternal border. The heart tones were normal. There were a few crepitant râles

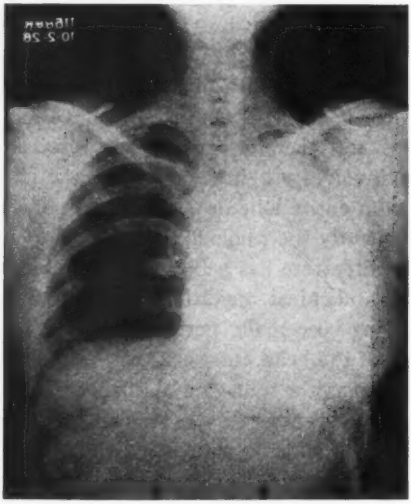

Fig. 7.—Film taken on entrance to hospital. Note the uniform density of entire left lung field except the extreme apex.

heard at the left apex. Breath tones were diminished at the apex and absent over the rest of the left chest except posteriorly just to the left of the spine, where there was bronchial breathing. Fremitus was absent except in the same region, where it was increased. The right lung findings were normal.

x-Ray examination by Dr. Raymond Householder: "The chest is symmetrical and well developed. Expansion of the right chest and excursion of the right leaf of the diaphragm is good. There is no movement of the diaphragm on the left side as seen

under the fluoroscope. The right lung field markings are interpreted as within the normal limits. The entire left lung field, except the extreme apex, is involved in a dense shadow. The fluoroscopy reveals a moderate deviation of the trachea toward the left. (Not noted on film.) The heart and mediastinal shadows are also displaced to the left, so that the apex lies at the left lateral chest wall. On the film the heart shadow is not identified as separate from the generalized density involving the entire left lung field. No definite lung markings are discernible on the film. The appearance is that of an atelectasis of the left lower lobe and the lower half of the left upper lobe with a marked displacement of the heart and lower mediastinal shadows to the left."

The patient was again examined by us October 6, 1926. There was no material change in the physical findings. The temperature had ranged from 99.2° to 98° F., pulse from 96 to 80, respirations from 26 to 22. A blood examination October 2d showed red blood-cells 5,100,000; white blood-cells 21,000. There was a definite polymorphonuclear increase seen on examination of a stained blood-smear. Blood-pressure was 90 systolic and 62 diastolic. The urine was negative.

Because of the marked flatness at the base, fluid was suspected, and Dr. Hutchison inserted a small trocar, but no fluid was obtained. During this procedure some air entered the pleural cavity. On returning from the operating room it was noted that the patient seemed to breathe more easily and was more comfortable. The pulse became slower, ranging from 72 to 65, and the respirations not exceeding 22 during the remainder of her stay in the hospital. The heart had returned to its normal position. The lung findings were those of a pneumothorax. Two days later there was found to be a collection of fluid at the left base. *x*-Ray examination at this time revealed a hydropneumothorax of the left side with a completely collapsed left lung. The heart and mediastinal shadows were in a normal position. There was fluid free in the pleural cavity up to the anterior end of the fourth rib and air above the free fluid.

October 20th another check-up fluoroscopy showed a return

of the lung to the chest wall with a very slight amount of fluid in the left costophrenic angle. The patient was discharged from the hospital October 24th, but returned for examination after two months, at which time there was no evidence of lung pathology, either upon physical or *x*-ray examination. The mechanism of massive collapse of the lung is not clear. It was attributed to a paralysis of the diaphragm, usually postdiphtheritic, by Pasteur[3] in 1890. He did not believe it due to obstruction of

Fig. 8.—Film taken six days later, two days after exploratory puncture. The fluid level is at the fourth rib anteriorly, and the left upper lobe margin is seen just to the left of the left sternal border.

the bronchi. Eliott and Dingley[4] showed that there was no displacement of the heart in cats after hemisection of the cord with resulting paralysis of one-half of the body. They cited the work of Lichtheim, who produced atelectasis experimentally by plugging a bronchus. They advanced the hypothesis that the collapse is due to an obstruction of a bronchus with absorption of the imprisoned air. Jackson and Lee[5] have demonstrated a mucous plug in a bronchus in several of their cases and have re-

lieved symptoms by removing the plug through the bronchoscope. Scott[6] in 1925 reviewed the literature and analyzed all the cases which were reported in sufficient detail, and added 4 cases. He feels that it is due to some reflex mechanism which produces either directly or secondarily by secretion an obstruction to the air passages supplying part of the lung. The rapid change in physical signs observed in 2 of his cases, where a marked change was noted within five minutes and thirty minutes respectively, would suggest a reflex mechanism. He suggests three possibilities:

1. Dilatation and stasis in blood-vessels could produce obstruction in bronchioles with outpouring of secretion.

2. Bronchial spasm suggests itself as a possibility on account of the sonorous râles found which vary from minute to minute. However, bronchial spasm as observed in asthma usually produces an emphysema instead of collapse.

3. Swelling of mucous membranes similar to an angioneurotic edema. He cites Hoover's work on the mechanism of anaphylaxis in guinea pigs, where he has shown that the bronchial obstruction is due to a swelling of the mucous membrane and not to spasm. After the initial obstruction the secretions undoubtedly are a factor in maintaining the obstruction.

The observations of Sante[2] in a case where the inflation was observed under direct fluoroscopic vision would support the theory of a reflex mechanism, as would the sudden onset of symptoms and the occurrence of contralateral collapse.

Bradford[1] studied the subject as occurring in connection with war wounds and feels that the mechanism is obscure. He mentions both views, but states that there are definite difficulties in accepting either. He suggests that it may be due to a temporary paralysis of the inspiratory mechanism of reflex origin. He states that the lung is shrunken, heavy, leaden blue in color, and airless. Massive collapse of a whole lung may occur without any evidence of any gross obstruction of the bronchus.

In view of these pathologic findings, the bronchoscopic findings of Jackson and Lee, and the clinical findings reported by others it would seem that there might be several factors involved,

but a reflex mechanism would seem to offer the most satisfactory explanation.

Massive collapse occurs at any age, but is most common in middle life, probably because of the greater number of operations during this period. Griffith[7] reports its occurrence in a child three weeks old.

Previous condition apparently has no definite bearing, as it occurs often in previously healthy individuals. Most cases reported in civil life have occurred after abdominal operation, especially appendectomies and herniotomies. Bradford[1] states that probably 5 to 10 per cent. of chest injuries were followed by massive collapse.

Massive collapse may occur without operation or injury. Hunt[8] reports a case of acute cholecystitis where massive collapse occurred while the patient was in the hospital awaiting operation. Regan[9] reports a case following poliomyelitis.

The onset of symptoms may be sudden, usually within forty-eight hours, one-half of the cases within twenty-four hours, according to Scott.[6] The symptoms at the onset may vary, but usually there is a rise in temperature with increase in pulse rate and respirations, which may or may not be associated with cough and pain in the chest. The temperature need not be high. Cyanosis may be the only symptom at the onset. The cough may be non-productive at first, but later there may be rather thick, tenacious sputum not blood-streaked or rusty. Symptoms may be almost entirely absent and the condition discovered incidently during examination of the chest.

Bradford[1] states that dyspnea is the most constant symptom and may vary greatly and is greatly increased by any exertion. Massive collapse may involve a whole lung without urgent symptoms, especially in the contralateral variety.

The physical signs differ from all other postoperative pulmonary complications, in that the heart and mediastinum are displaced to the affected side. The extent to which the heart is displaced varies, but is usually marked. The apex-beat is displaced upward and may reach the third rib. There is usually retraction of the affected side and limited or entire absence of

expansion. The percussion note is dull, may be flat, especially at the base posteriorly. The diaphragm is high and immobile on the affected side. Breath tones are either greatly diminished, may be absent, or there may be bronchial breathing. This may vary from minute to minute, as observed by Scott.[6] Usually the change is from diminished breath tones to bronchial breathing, but the reverse may occur.[1] Râles may be absent during the entire course, but may be present. They are more commonly present during the later stages when the lung is re-expanding.

The duration is very variable. The signs may clear up in a few days or even in a few hours. Scott[6] cites a case reported by Leopold with alternate collapse and re-expansion on successive days over a period of six days. This was verified by *x*-ray. Usually there will be complete restoration to normal in three to four weeks.

The prognosis is good unless complications arise. The most common complication is pneumonia, but pleurisy with effusion may occur.

The treatment of massive collapse is dismissed by Bradford[1] with a few words. He suggests that the patient be kept in a position which will encourage free movement of the chest. Scott[6] suggests morphin to control the cough and pain. He tried atropin and epinephrin, but without result. He states that the patient will usually lie on the affected side. Lying on the unaffected side causes dyspnea. Blow bottles have not been of any benefit and might increase the discomfort by overinflating the sound lung, increasing the displacement of the heart and mediastinum.

The apparent relief which followed the entrance of air into the pleural cavity suggested to us the advisability of producing a pneumothorax in the treatment of unilateral massive collapse with marked dyspnea, tachycardia, or cyanosis. On reviewing the recent literature we found that Farris[10] has advocated this procedure as treatment in cases with marked symptoms, and cites several cases where it was followed by definite and almost immediate relief. The trocar must be used with some degree of caution because of the marked displacement of the heart and

the density of the collapsed lung, as the possibility of cardiac puncture must not be overlooked. However, we feel that the results reported by Farris and the rather striking change noted in our case, justifies its use when marked cyanosis, dyspnea, tachycardia, or distress are present.

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CLINIC OF DR. JAMES H. HUTTON

CHICAGO

ENDOCRINE DISORDERS AS RELATED TO MORE COMMON SYNDROMES

COMMON sense furnishes a good guide to action in this field. Consider that every normal human being has, roughly, twelve endocrine glands. That the pituitary, adrenals, and pancreas, and probably the parathyroids are essential to life—not that life cannot be maintained without all of them, but without any one of them. The thyroid is necessary to our normal physical and mental development and to the normal exercise of mental and physical functions throughout life. The normal function of the gonads is necessary to the propagation of our species.

Does it stand to reason to suppose that all of these glands are enclosed in some sort of fool-proof cage and so escape damage inflicted on other parts of the body by the infections and other strains, physical and mental, incident to modern life?

Is it not reasonable to conceive that the disturbance of function should be reflected in symptoms?

I would like to combat three common misapprehensions regarding endocrine disorders. First, that they are strange, rare, or peculiar syndromes. Second, that when these disturbances do occur the victim is marked by some unusual and striking physical peculiarities, and, finally, *that after a diagnosis is made nothing can be accomplished by endocrine therapy.*

Regarding misapprehension number one, that they are strange, rare, or peculiar syndromes. Consider that there are about 500,000 diabetics in the United States. Perhaps it has never occurred to you that diabetes mellitus is an endocrine disorder; but please remember that one of the things we know most certainly about this disease is that the internal secretion of the

pancreas is supplied to the body in insufficient quantities. There is no way of knowing how many goiters there are, and for every goiter there are probably 3 cases of hypothyroidism. Every woman who reaches the menopause has some endocrine upset, the severity of this upset depending on circumstances with which we are not now familiar. Many menstrual disturbances, occurring either at puberty or in later life, are due to ovarian insufficiency, and the nausea and vomiting of pregnancy probably are expressions of endocrine disturbances of one kind or another.

Misapprehension number two. That when endocrine disorders do occur the victim is marked by some unusual and striking physical characteristics. As a matter of fact, these things are true in unusual, neglected, or severe cases. The pituitary giant, the acromegalic, the youngster with a teratoma of the pineal, offer examples of this type of case. Obesity of thyroid or pituitary distribution and the changes occurring in connection with a tumor of the adrenal cortex offer other examples of this conception of endocrine disturbances and their results.

Confining our diagnosis of endocrine disorders to the recognition of such cases would be exhibiting the same diagnostic acumen in this field as would the recognition of cancer of the breast when the lymphatics are involved and metastases have occurred, or with the recognition of diabetes after the onset of acidosis.

Like these more common conditions, endocrine disturbances should be recognized earlier or when the condition is of milder type.

The prognosis of most endocrine disorders is good in proportion to the earliness with which they are recognized and the promptness and thoroughness with which treatment is instituted and carried out. A mild hypothyroidism is much more easily cured and the damage to the patient is more easily repaired than if it goes on to well-marked myxedema of some years, standing before correct treatment is instituted.

Endocrine disorders accompany, either as sequelæ, complications, or as a matter of coincidence, many common disorders, without producing marked changes in the physical appearance of the patient.

Chronic diseases furnish frequent examples of how endocrine disorders complicate more commonly recognized syndromes. Many cases of arthritis have an endocrine factor of importance. Many cases of "chronic appendicitis" are better treated by endocrine therapy than by appendectomy. Many times the complaints which lead to pelvic operations are due to endocrine disorders instead of lacerations of the cervix or perineum or the small ovarian cyst.

Regarding misapprehension number three, that after a diagnosis is made nothing can be accomplished by endocrine therapy. As an example of the efficacy of endocrine therapy think of insulin in the treatment of the severe acidosis of diabetes mellitus or the severe diabetes without acidosis. Desiccated thyroid or thyroxin, preferably the latter, is a specific for myxedema and the milder grades of hypothyroidism. The favorable effects of ovarian medication either by mouth or subcutaneously are well known in the treatment of ovarian insufficiencies, either those of the menopause or earlier life; not to mention the many cases of dysmenorrhea so easily controlled by this simple medication, even some severe cases that have been curetted, dilated, and tamponaded without avail. The nausea and vomiting of pregnancy sometimes responds more readily to corpus luteum medication than to any other treatment.

I want to present 3 case histories to illustrate the foregoing statements.

Case I.—Mrs. E. L. H., age seventy, referred tby Dr. Rutherford B. Irones of San Diego, California, first seen June 16, 1925. Her complaints, as given partly by the patient and supplemented by her sister, were as follows:

1. Precordial distress and fear of angina pectoris and death. Her entire attention was centered on her heart.

2. Weakness so extreme she could hardly stand and could not walk without assistance.

3. Some pain in her legs, present most of the time.

4. Loss of memory for recent events and partially disoriented as to time.

5. Difficulty in talking. Her speech was thick and at times slurring.

Past History.—About 1918 she began to gain decidedly in weight, and in the course of the next three or four years gained up to about 170 pounds from a normal of about 135 pounds. This fact is significant in view of her age, as a gain in weight is abnormal at this time of life. During this period she was easily fatigued and was tired most of the time. She had precordial pain occasionally.

She was examined a number of times and told her "metabolism was not right." A basal metabolic determination was not made. A dentist extracted her teeth without relieving her complaints in any way.

She suffered a severe carbon-monoxid poisoning in May, 1923. Following this she was unable to walk for some weeks and was quite ill for some months. However, she eventually regained her usual health and was getting along quite well until the late summer of 1924, when she had "influenza." Some time after this she consulted Dr. Irones, whose statement may be summarized as follows:

"I saw Mrs. H. first on September 3, 1924. I will not go into an elaborate detailed report of this examination, but just briefly state that the essential points were as follows:

"It seems that about eighteen months previous to her visit to me she suffered a severe carbon-monoxid poisoning. I sent her to Dr. H. A. Thompson for a blood-count, which showed a marked secondary anemia, the reds being 2,904,000, whites 6500, hemoglobin 60 per cent., color index 1, a slight poikilocytosis, microcytes few, macroytes few, mononuclears 13 per cent. Urine was negative.

"I gave her iron and red marrow at the time, and by September 26th her hemoglobin was increased to 80 per cent. (Dare).

"On October 22, 1924 another blood-count was made by Dr. Thompson, showing reds 4,664,000, whites 6700, hemoglobin 70 per cent. There was, however, evidence of a multiple neuritis. She tired easily and had some vague pains in the legs, but all of this cleared up by December, 1924, when she was taken with a

severe attack of influenza. From that time on the leg pains and tired feeling and some in-coördination in walking and a loss of almost entirely all knee-jerks was noted. This continued on and off until March 15, 1925, when she had a rather marked attack of angina pectoris. This I feel was not a pseudo attack, for the pains were typical. The blood-pressure at that time was systolic 190, dropping to 60 diastolic, leaving a pulse pressure of 130. The pulse rate was 138.

"She had several of these attacks which responded very nicely to the nitrites, but the leg pains increased—the knee-jerks became completely obliterated and I insisted upon her becoming an absolute bed case.

"On March 17th she had an attack of syncope, the blood-pressure dropped to 120 systolic. The radial pulse ran up to 160 and the patient gave a picture of cardiac decompensation. She rallied from this, however, and had several other attacks of precordial pain. I had her removed to a private sanitarium, where she did very well up until May 20th, when the first sign of Korsakoff's psychosis presented itself. This was fairly typical and was evident on and off until the time she left San Diego for Chicago.

"There has been no loss of bladder reflexes at any time, although we did have some considerable trouble with constipation, but after entering the sanitarium this condition soon regulated itself so she had daily bowel movements without any aid.

"A true diagnosis of Mrs. H.'s case has never been communicated to her personally because of the fear of an adverse psychologic effect on her part."

Our examination, begun June 16, 1925, showed the following:

Temperature 97, pulse 58, blood-pressure 140/90. The skin was not especially dry, but was very pale, a malar flush was present. The outer half of the eyebrows were entirely missing. The body hair suit was very scant and the axillary hair was almost entirely gone. The nails were striated, but not markedly brittle. Most of the teeth were false. The throat was negative, as was also the nose and accessory sinuses. The pupils were regular, equal, and reacted to light and accommodation. The fundi were normal in appearance. The reflexes were sluggish,

but otherwise normal. The Romberg was positive, perhaps because of her weakness; co-ordination was fair. The heart was of normal size, the sounds of fair quality, and no murmurs could be heard. Its response to exercise was not tested. The lungs were clear. The abdomen revealed no masses, no area of tenderness, and the liver, spleen, and kidneys were not palpable.

Dr. George W. Hall who saw her in consultation decided that no abnormality of the nervous system existed except such as might be secondary to her general condition.

Laboratory findings were as follows:

The urinalysis showed no sugar, no albumin, few hyaline casts, numerous epithelial cells. The blood Wassermann test was negative.

Blood-count showed:

Hemoglobin	70 per cent.
Red blood-corpuscles	3,580,000
White blood-corpuscles	5,300

Blood chemistry showed:

	Mgm. per 100 c.c. blood.
Sugar	114
Non-protein nitrogen	24.57
Calcium	12.2
Uric acid	3.15
Creatinin	1.65

The basal metabolic rate of minus 33 per cent. verified the diagnosis of atypical myxedema.

Treatment.—She was given 4 grains of desiccated thyroid three times a day. In less than a week she decided to leave the city, but explained that she would be under the immediate observation of a physiscian. On this understanding she was allowed to continue the thyroid in the dosage mentioned. A letter explaining the situation was sent to the doctor.

Within a few weeks her weakness increased and her mental faculties declined to the point where she failed to recognize members of her immediate family. These symptoms cleared up when the dose of thyroid was cut down. She improved consider-

ably after that, and on her return to the city in September was markedly improved. She was, however, very nervous and her pulse was 100. Her basal metabolic rate was found to be plus 40 per cent. Thyroid was entirely stopped until the rate dropped to minus 10 per cent. She was then given 10 mgm. of thyroxin intravenously. Her response to this was no better than to thyroid, so that she was placed on that a little later. Eventually it was determined that 1 grain of thyroid three times a day would maintain her basal metabolic rate near normal and she is kept on that dosage.

Her improvement was rapid, so that by December 1, 1925 she could indulge in the activities of the average woman of her age. That condition has remained up to the present time.

In this case the endocrine factor was the key to the whole situation and undoubtedly explains the difference between the findings of Dr. Hall and Dr. Irones, as thyroid deficiency many times produces symptoms that justify a diagnosis of multiple neuritis and sometimes of tabes.

As in most conditions the history was of great value, showing that the hypothyroidism began several years before her monoxid poisoning. In this case the more common syndromes, at least ones with which we are more familiar and which have a more respectable standing in medical literature and nomenclature, were well marked and correctly recognized by a man of unusual professional attainments, so that the diagnosis cannot be questioned. And these common syndromes pretty well masked the underlying primary condition.

Case II.—Mrs. C., age thirty-three, referred by Dr. J. V. Fowler, December, 1924. Her complaints were listed as follows:

1. Headache occurring after every meal. This has been present for years.

2. Sense of substernal pressure and inability to breathe. This occurs or is worse when she is in a crowd or is nervous.

3. Tachycardia occurring at the same time and under the same circumstances as the substernal pressure.

4. Pain and a burning sensation in the right lower quadrant

of the abdomen. This began at the age of fourteen, about the time of her first period. It is present most of the time.

5. A great deal of belching. Immediately after breakfast she has pain in the right upper quadrant and is nauseated, but does not vomit. This recurs after every meal, but is worse after breakfast than at any other time.

6. She is afraid to go out alone and is afraid of a crowd.

7. She is blue and depressed, with thoughts of suicide, but no serious intentions along that line.

Past History.—A head injury at the age of eighteen or nineteen. She had headache for a week afterward, but was not unconscious at the time and does not remember that polyuria occurred. Tonsillectomy. One miscarriage induced.

She has been under medical supervision most of her life, as she has never been well. The diagnoses have ranged from toxic goiter to neurasthenia. Dr. Fowler stated that he was afraid she would become mentally unbalanced in a short time.

Family history is of no significance.

Developmental History.—She knows little about her weight at birth, time of walking, talking, etc., but is sure that as a baby she was underdeveloped and was "starved." Her progress in school was very slow.

Menstrual History.—The first period occurred at fourteen. This was followed by an amenorrheic period of thirteen months. She has always been irregular, the periods occurring six weeks to three months apart. The flow lasts four days and is not painful. She does not know that her complaints are either better or worse during the flow.

Habits.—Appetite fair, does not sleep well, bad dreams, constipated.

Examination.—Height 5 feet, weighing 122 pounds, temperature 98° F., pulse 100, blood-pressure 110/80.

The fingers are short and tapering, the palms are broad as compared with their length, the wrists are relatively broad. There is no padding on the dorsum of the hands or feet nor about the wrists and ankles, nor in the supraclavicular area. Marked tremor of the hands and tongue. The skin is not dry. The nails

are not ridged or brittle, the hair is not markedly dry, but the hair suit is rather scant. The teeth are regular and of fair quality. The tonsils are out. The nose is negative. The pupils are equal, regular, and react to light and accommodation, the fundi are normal. There are no palpable glands. The thyroid is very small. The heart is negative except for the rate. The lungs are clear. The abdomen is generally tender, but no masses can be felt, and neither the liver, spleen, nor kidneys are palpable. The tenderness was rather more marked over the lower right quadrant.

The laboratory findings were as follows:

Basal metabolic rate plus 9 per cent. Non-surgical drainage of the gall-bladder showed normal bile. Blood Wassermann was negative. Blood chemistry was reported as follows:

	Mgm. per 100 c.c. blood.
Sugar	82.70
Non-protein nitrogen	50.00
Calcium	7.5
Uric acid	5.00
Cholesterol	130.00

Stomach contents showed no abnormalities.

Blood-count showed:

Red blood-corpuscles	4,230,000
White blood-corpuscles	6,800
Hemoglobin	90 per cent.

The Goetsch test showed a rise in the pulse rate up to 130 with a tremor, chilly sensation, and skin reaction the size of a silver dollar. The urinalyses were essentially negative except for low specific gravity. Renal function test was as follows:

One c.c. phenolsulphonephthalein given intramuscularly showed:

30 per cent. the first hour
25 per cent. the second hour
55 per cent. total

Quantity of urine:
400 c.c. first hour
50 c.c. second hour

Diagnosis.—Ovarian insufficiency, hypopituitarism anterior lobe, pre-adolescent as to time of onset, and a hypocalcemia. The diagnosis of ovarian insufficiency was based on the irregularity of the periods, the burning pain in the right lower quadrant of the abdomen, and the preceding pituitary insufficiency.

The diagnosis of pituitary insufficiency was based on the stature, the short tapering fingers, broad palm and wrists, the period of amenorrhea following the first period. The substernal distress was probably partly, at least, due to pituitary insufficiency.

Treatment.—She was first given ovarian residue, one ampule subcutaneously once a week. Five grains of the same three times a day per month. She was also given considerable doses of calcium in various forms. The amount of water was crowded. She was given some directions about overcoming her air swallowing habits. Later she was given anterior pituitary preparations by mouth and subcutaneously.

Progress.—Her response to treatment was prompt and satisfactory. Her belching promptly ceased and she has abdominal pain at infrequent intervals. Within two weeks she was sleeping better, dreaming less, and awakening feeling refreshed. Her tachycardia was slower in responding, but at the present time her pulse is about 80. The menstrual periods became regular within the next two months and have remained so with one or two exceptions. When the periods are delayed she feels badly, having some headache, tachycardia, and pain in the right lower quadrant of the abdomen.

Her fears have largely disappeared, so that she goes downtown alone and is able to entertain friends in her home, things she had not been able to do for over seven years.

Discussion.—This was an endocrine disturbance pure and simple, that is, there were few or no complicating features and the endocrine disturbances did not occur as complications or sequelæ to other conditions, but were primary and apparently congenital. Whether her pituitary insufficiency was primary and responsible for her "starved" condition in infancy and childhood we cannot tell certainly, but it was probably the thing most at fault.

Mrs. C. doubtless had her crippling endocrine ailment all her life. This shows how study of the endocrine system will many times furnish the key to a puzzling situation and help us to put the patient on his feet and restore him to a fairly normal place in the economic scheme of things. Whenever a chronic condition presents baffling symptoms it is especially apt to have an endocrine factor of considerable weight.

Case III.—Mrs. L., age fifty; chief complaint, arthritis involving the distal joints of all the fingers. The knees were also slightly involved. The condition had been present in the fingers for five years, but the knees had only recently shown any changes.

Her general health was good and she had been told by her doctor that nothing could be done for the arthritis. There had been no change in her weight in recent years.

Habits.—Her appetite was very good, but she had recently cut down her food intake, especially the sugars and starches. Her bowels were regular, but for twenty years she had to use cathartics. She drank very little tea and coffee and no alcohol or cocoa.

Past History.—Migraine, beginning at the age of twelve or thirteen, having no relation to the menstrual periods. It occurs much less frequently now and is less severe, coming on about three times a year. Bell's palsy at the age of thirty-four. She feels that she has always had a tendency to rheumatism. Tonsils were removed two years ago. She has had a good deal of inflammation in her mouth, with many aphthous patches on the tongue and palate.

Family History.—She was married late in life and has never been pregnant. None of her relatives are subject to arthritis.

Menstrual History.—The first period occurred at fourteen, followed by an amenorrheic period of six months. The periods then became regular, occurring every twenty-eight days and lasting four or five days without pain. They stopped about six months before she came under observation. This cessation was not accompanied by any circulatory or nervous phenomena.

Examination.—Temperature 98° F., pulse 88, blood-pressure 140/80. The distal joints of all the fingers were swollen. Some of them were painful to touch and appeared to have become inflamed recently (as indeed they had), while others appeared to have been in their present condition for a long time. The nails were ridged and brittle. There was some creaking in the knees, especially in the right.

The skin was not especially dry or did the hair seem dry and brittle. The pubic and axillary hair was rather scant, but the outer third of the eyebrows was not thin. There were no palpable glands and the thyroid was very small. The tongue was coated; the teeth appeared in good condition, and the *x*-ray confirmed this appearance. The nose was normal. The pupils were equal, regular, and reacted to light and to accommodation, and the fundi were normal. The reflexes were very active; the Romberg sign was negative, but co-ordination was poor. There was a slight deposit of fat about the ankles, but there was no padding on the dorsum of the feet or hands, about the wrists, or in the supraclavicular areas.

The heart was of normal size and in normal position and gave a good response to exercise. No murmurs or adventitious sounds were heard. The lungs were clear, the abdomen was negative in that the liver, spleen, and kidneys could not be palpated and no masses could be felt.

Laboratory Reports.—Urinalysis showed no sugar; a faint trace of albumin; occasional finely granular casts; numerous pus-cells. The stool showed no parasites or ova.

Blood chemistry showed:

	Mgm. per 100 c.c. blood.
Sugar	105
Uric acid	4
Urea	17

Blood-count showed:

Hemoglobin	70 per cent.
Red blood-corpuscles	4,700,000
White blood-corpuscles	5,100

Differential white count:

Polymorphonuclear neutrophils	53
Polymorphonuclear eosinophils	6
Lymphocytes	30
Large mononuclears	11

The basal metabolic rate was minus 22 per cent.

The only significant thing about the blood-count was the eosinophilia. This condition is quite frequently encountered in myxedema.

Treatment.—She was given ½-grain thyroid three times a day, with some salicylates to control the pain. The diet was made quite low in carbohydrates. After a short time hot flashes developed, and 5 grains of ovarian residue three times a day was added to the thyroid. The salicylates were then stopped. By the last of January there was definite improvement in the arthritic condition, which has continued satisfactorily to the present time. (She came under observation December, 1925.)

The blood-count on March 18, 1926 showed only 3 per cent. eosinophils and the basal metabolic rate on that date was exactly normal.

Mrs. L. is typical of another phase of this question. She had a well-established arthritis. Because no focus of infection could be found her doctor felt that nothing could be done in the way of treatment except "watchful expectancy," probably hoping the case would not develop into a crippling deformity and being ready to take active measures to correct the deformity should any occur.

No focal infection could be demonstrated after the most painstaking search, and the fact that, to say the least, other factors were operative as etiologic factors is proved by the results of treatment. A well-marked ovarian insufficiency and a pronounced thyroid deficiency furnished the basis for treatment which resulted in a marked and satisfactory improvement. The progress of the condition was stopped and there was retrogression of the condition in some of the joints.

This shows how the recognition of an endocrine disorder as

part of a patient's trouble may help us to cure the condition of which he complains, or, at least, to stop its progress and change a therapeutic campaign from hopelessness to success.

Conclusions.—Endocrine disorders are of common occurrence.

The severe cases or neglected ones are marked by striking physical characteristics. The milder cases and those most frequently seen are not so marked, but such physical markings as are present are those of the severe cases in lesser degree.

Endocrine disturbances occur most frequently as complications or sequelæ to more commonly known conditions.

The same attention should be paid to the function of the endocrines as is paid to the condition of the gastro-intestinal tract or the heart or kidneys. Considerable criticism is directed at the man who has charge of a case for any length of time if he fails to make an analysis of the urine. Most of us feel that that should be a routine procedure in every case regardless of the nature of the patient's complaints. The same attention should be paid to the endocrine system.

A careful history is of as much importance, if not more, in endocrine disorders as in other conditions.

Hypofunction of any of these glands is much more amenable to treatment than is hyperfunction. For the former condition we have specific medication in the form of substitution therapy.

The most that any endocrinologist can ask is that the profession recognize the endocrine glands as part of the body, having important functions to perform and giving rise to various symptoms when these functions are not properly performed. In other words, to familiarize himself with such facts as we have concerning these interesting organs and investigate their condition as carefully as possible when examining any patient. Just as one goes into the question of the condition of the bowels or the kidneys or the sort of diet the patient lives on.

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FUSOSPIROCHETAL PULMONARY ABSCESS

THE term "fusospirochetal pulmonary abscess" is introduced to cover what we believe is a common type of a suppurative lesion in the lung. The majority of abscesses characterized by foul expectoration when investigated bacteriologically are of this type. It is our purpose to bring out the condition as a clinical entity worthy of special consideration, as our studies of the bacteria responsible for the process suggest additional therapeutic agents. In previous work[1] we brought out the important rôle that the anaërobes, particularly the fusiform bacillus and the symbiotic spirochetes, play in the development of suppurative and gangrenous lesions of the lung. Since that time we have encountered additional cases which support our views and also bring out our contention of the great importance of the underlying predisposing factors. An analysis of the cases that will be cited will illustrate how often the underlying factors may be obscure, and that often the progress of the patient will best show the associated conditions.

Lesions due to fusiform bacilli and spirochetes are commonly seen and recognized in the mouth by general practitioners as well as dental and oral specialists. The tonsillar and pharyngeal lesions in the form of Vincent's angina is usually an acute state, but may become subacute or chronic with the development of an ulcerating lesion and foul odor. Similar conditions are commonly seen about the teeth. It is, however, surprising how few

[1] Pilot, I., and Davis, D. J.: Studies of Fusiform Bacilli and Spirochetes; IX. Their Rôle in Abscess, Gangrene, Bronchiectasis, Arch. Int. Med., 34, 1924, 313–354.

pulmonary lesions have been identified with these bacteria. These anaërobes normally reside in most persons about the teeth in the tartar and often in the tonsillar crypts, where they may be found in great numbers in the firm globular masses that plug the openings of the crypts. It therefore should not be unusual that under certain conditions these anaërobes, like the streptococcus and pneumococcus, find their way into the lower respiratory passages and give rise to definite lesions. Our first studies led to 37 cases, and would indicate that infections with these anaërobes in the bronchi and lungs are not uncommon. The sputum of the patients with lung abscess invariably reveals a mixture of bacteria, but when the odor is foul the anaërobes will be found in considerable numbers. The relationship of the associated bacteria to the anaërobes will be discussed later.

Let us examine some of the predisposing factors that are so important in the development of fusospirochetal abscess. We have noted that the lesion develops in patients depleted generally by acute or chronic ailments, or complicates a pathologic state in or about the respiratory passages.

Acute respiratory infections, such as pneumonia, influenza, bronchitis, are rarely complicated by such abscesses. Many instances of an abscess without an outstanding etiologic factor are said to be preceded by a cold or a bronchitis in the history given by the patient. An analysis of such histories will show that the symptoms are not independent of the pulmonary abscess, but are part of the early manifestations of the lung lesion. In patients whose abscess develops with an onset like that of an upper respiratory infection it is well to bear in mind that other factors may be underlying the condition.

Diabetes often combined with other factors lead to pulmonary abscess of the putrid type. In one instance a gangrene complicated a lobar pneumonia in a young diabetic. In another the lesion was superimposed upon a bronchiectatic cavity containing an aspirated tooth for a period of thirty years. The lesions associated with diabetes tend toward multiple abscess formation with marked necrosis and gangrene.

Local disease in the upper respiratory passages are sometimes

outstanding features. About diseased gums and teeth the anaërobes are found in great bundance. In patients undergoing anesthesia these sites may be the sources of infection. In the tonsils where the anaërobes occasionally occur in large numbers we have an important source of infection when tonsillectomy is performed. Of special interest illustrating that aspiration is a common mode of infection is the development of an abscess or gangrene of the lung as a complication of carcinoma of the larynx or of a perforating lesion of esophageal carcinoma. In carcinomata of these regions we have demonstrated the same anaërobes in large numbers in the necrotic ulcers.

Various pulmonary conditions may become complicated by fusospirochetal abscess, and these conditions should be looked for when an abscess appears without any outstanding etiology. Most important is the presence of an old abscess cavity or the presence of bronchiectatic cavities. It seems that the anaërobes become easily established as saprophytes and may be demonstrated in the sputum when the sputum is not foul. When patients with bronchiectasis develop fever, chills, and profuse foul expectoration the bacteria increase in numbers and at autopsy may be demonstrated in newly formed abscesses. Bronchogenic carcinoma may be complicated in a similar way. Tuberculosis may be associated and often may be masked by an abscess (Case I). We have observed instances where in one tuberculosis, bronchiectasis and abscess, in another carcinoma, bronchiectasis and abscess were associated in the same lung.

Of outstanding importance is the rôle of general anesthesia in the production of the pulmonary complication. Not uncommonly the abscess follows tonsillectomy. Two views have been held as to the mode of development of post-tonsillectomy abscess, one the inspiration and the other the embolic theory. In the latter one requires a thrombosed vein with infected emboli lodging in the parenchyma of the lung. Such may be the route in a few cases, but it is doubtful if a mixture of bacteria consisting of pyogens and the anaërobes could all be carried by the blood-stream. The metastatic abscess of the brain from bronchiectasis reveals bacilli and cocci, but no spirochetes, although

the latter may be present in the lung. In favor of the aspiration theory is a striking similarity of the anaërobes about the teeth and tonsils with those found in the abscess; the frequency of the abscess close to the hilum with often sudden rupture into a bronchus; the demonstration by bronchoscopy (McGinnis) in early cases of a disintegrating blood-clot of foul odor in a bronchus. Furthermore, the postoperative pulmonary abscess following a clean abdominal or hernia operation contains the same anaërobes of the mouth. Of further interest in this connection is the association of fusospirochetal abscess with foreign bodies in the bronchi. In one instance a piece of wire snare was demonstrated; another expectorated a piece of cancellous bone, and a third had a tooth in a bronchiectatic cavity.[1] One cannot deny that the embolic route may not be a mode of infection, for we have observed one instance of a hemorrhagic infarct becoming infected with the anaërobes, but from our observations this mode is exceptional.

Of peculiar interest is the development of a fusospirochetal abscess without any apparent cause. Our previous experience has taught us that if the underlying cause is not ascertained, the subsequent course or the autopsy may yield the associated state. As pointed out in another connection, a bronchiectasis, tuberculosis, carcinoma or a foreign body may be demonstrated. In one recent case we were unable to find any associated pathology (Case VI).

In putrid pulmonary abscesses the fusiform bacillus and its symbiotic spirochetes with few exceptions is the cause of the odor. But associated with these anaërobes are pyogenic bacteria which may play an important rôle and may even be responsible for the course and termination of the abscess. Recent writers on spirochetal pulmonary infections have either failed to recognize these pyogens or have preferred to neglect their importance. The bacteria commonly encountered are the Streptococcus hemolyticus, Streptococcus viridans, pneumonococcus, Micrococcus catarrhalis, staphylococcus, Bacillus influenza, and others. The streptococci are most constantly present. It is noteworthy that when the Streptococcus hemolyticus is present the condition

is more serious and the outcome often unfavorable. Within the lesion the bacteria often have a characteristic distribution. The necrotic wall of the abscess cavity reveals a mixture of bacilli, spirochetes, and cocci. The spirochetes may be seen at the margin advancing into the surrounding tissue, while the cocci may be observed in the most remote areas of consolidation. This distribution suggests that the cocci are first concerned in producing consolidation and that the anaërobes find these involved areas excellent soil for the production of a necrotic lesion. This tendency to necrotic abscess formation extends and involves adjacent areas while the cocci advance to new regions. The pyogens and the anaërobes undoubtedly influence each other and establish a symbiotic relationship in the lung tissue. If either group of organisms can be affected the progress of the lesion can be changed. It is upon this basis that salvarsan is administered in our patients in the hope of influencing the fusiform bacillus and the spirochetes and permit the pyogenes to be acted upon by the immune bodies of the patient.

The lesion of uncomplicated fusospirochetal abscess is that of a bronchopneumonic consolidation with central necrosis. The process is at first acute and often becomes subacute and chronic. Any lobe may be involved, but the most common site is the right lower lobe. Severe lesions may be multiple from the beginning or there may be rapid spread from the primary abscess with the formation of new abscesses. In this spreading type of lesion there is a great tendency to gangrene and the abscess cavities show marked necrosis with green discoloration and intense putrid odor. The adjacent portions of the lungs are involved in a bronchopneumonic process usually of the interstitial type. The pleuræ may become involved either in the form of a serofibrinous pleuritis due to the pyogens or a putrid empyema as the result of the extension of the necrotic process of the pleura. In the putrid pus all the bacteria concerned in the development of the abscess may be found. In chronic abscesses the wall becomes thickened due to fibrosis. The tendency to healing in such lesions is slow, and when the necrosis disappears the abscess cavity may remain lined by granulation tissue surrounded by the

fibrous wall. In such lesions there is often a tendency to recurrent acute inflammation.

The symptoms are variable, particularly in the complicated abscess. The acute form gives fairly constant symptoms of cough, fever, perhaps with chills and often chest pain. The fever varies from a slight elevation to as high as 104° F. Leukocytosis is present. The symptoms continue without abatement until the tenth, twelfth, or fourteenth day, when the sputum may gradually or suddenly appear as a foul-smelling expectoration. In sudden rupture the amount is copious and is often followed by a drop of temperature with rapid resolution and recovery. When expectoration is not sudden or profuse the condition is apt to become subacute or chronic, with continued fever, leukocytosis, progressive weakness, and other evidences of sepsis. The sputum remains foul often for an extended period of weeks or months. Acute abscesses that resolve quickly, as a rule, do not recur, but in the subacute or chronic types periods of improvement when the sputum is reduced, fever absent, often alternate with periods of exacerbations. When the abscesses are multiple from the beginning the course is either rapidly unfavorable, with a picture of marked sepsis, or the condition becomes chronic with no tendency to improvement.

The physical findings depend upon the location of the abscess. Single abscesses that are deep seated or at the hilum may give indefinite findings. Large abscesses usually give signs of consolidation at first limited to impairment of resonance with tubular or more often bronchovesicular breathing. Râles appear later or when the foul expectoration develops. As the lesion is often far within the parenchyma of the lung, signs of cavity formation may be lacking. The findings may be obscured by the presence of exudate in the pleural cavity.

The *x*-ray is of great value in localizing the lesion and furnishes us with the best means of studying the course of the process. In the single abscess the shadow appears fairly well defined and later presents a central clear area of cavitation. In the favorable cases the shadow representing the pneumonitis gradually disappears, the cavity diminishes in size, and only a faint in-

filtration may remain as residuum. In the progressive cases the outlines of the shadow are fuzzy and the margins spread to previously clear areas. The cavity becomes larger or multiple cavities may appear. In the more chronic cases the cavity is often large and contains a fluid level. The surrounding density is not as marked as it is in the acute stage.

Complications that arise are chiefly referable to the pleura. Extension of the infection may occur, resulting in the development of serofibrinous or a putrid empyema. In the former type the bacteria found are the pyogens, especially the Streptococcus hemolyticus; in the latter the anaërobes as well as the pyogens similar to those seen in the sputum are found in great numbers. A striking complication is the formation of a metastatic brain abscess in the cerebral hemispheres. The pus is putrid and contains pyogens and the fusiform bacillus, but no spirochetes in those we have studied.

The diagnosis can readily be made. The history of an etiologic factor and the development of cough, fever, chills, and foul expectoration are characteristic. In patients giving such symptoms if the physical signs are wanting the *x*-ray will reveal the abscess. The sputum provides one of the most important features, and its study will determine whether the lesions are due to the fusospirochetes. The daily twenty-four specimen should be collected and the quantity measured. On standing the three layers form, an upper frothy, mildly viscid, and lower purulent. Early, when the sputum first become profuse, the odor is very foul, and as the cavity enlarges and drainage becomes easier it may assume a peculiar sweetish character; if drainage is poor or periodic, the odor remains foul.

In the examination of the sputum certain precautions are necessary before the findings can be properly interpreted. The patient should be instructed to clean the mouth and brush teeth several times daily in order to reduce to a minimum the contaminating bacteria of the mouth. Before making smears or cultures the sputum should be washed in saline solution. This is performed by transfering with a wire loop small portions of the sputum, preferably from the bottom purulent layer, and carrying

the material through four dishes containing sterile physiologic salt solution. After washing, smears are made on the slide by gentle spreading. The smear should not be too thin and allowed to dry thoroughly in air before staining. These steps are necessary in order to preserve the morphology of the spirochetes which are easily distorted. For staining, 5 to 10 per cent. of freshly prepared aqueous fuchsin applied for five minutes gives good results. Gentian-violet may also be employed. The Fontana stain will bring out the spirochetes as intense brown or black spirals, but gives little information in regard to the associated flora. The sputum will reveal mixtures of bacteria, bacilli, cocci, and spirochetes. The bacilli are of various types, but those important are the fusiform, which occur as pleomorphic forms, either straight or curved bacilli with pointed ends. Cocci particularly are constantly present. The spirochetes are variable in their morphology, occurring as either long or short spirals, slender or coarse. If few are present a freshly collected specimen may reveal many. It is not at all unusual to find spirochetes only after repeated examinations. The fusiform bacilli however, are constant and their presence should be regarded as evidence that the spirochetes may be in the lesions. It was pointed out that the spirochetes may be most profuse at the margin of the abscess wall, while the bacilli appear in abundance in the necrotic lining. On several occasions we have noted the presence of large numbers of spirochetes in the lung at autopsy of a patient whose sputum during life revealed many fusiform bacilli and only few spirochetes.

The sputum of patients with abscess superimposed on other pulmonary lesions does not differ materially from the sputum of the uncomplicated. Tubercle bacilli should always be sought for to rule out an underlying associated tuberculosis. Blood is sometimes present and has lead to a confusion of these abscess cases with the hemorrhagic bronchitis of Castellani. This type of bronchitis has been described in the tropics and is characterized by a thin, watery, pink odorless sputum which contains the Spirochetæ bronchialis. The process is apparently limited to

the bronchi and tends to spontaneous improvement or rapid clearance by the use of salvarsan or sodium cacodylate. The reports, however, of subacute or chronic forms of bronchitis associated with spirochetes are, in our opinion, similar to our cases of fusospirochetal abscess. An analysis of such reports will show that the patients have putrid sputum and that fusiform bacilli and cocci are also present, but their relationship to the lesion is not recognized.

Of particular interest is the diagnosis of obvious cases of lung abscess as syphilis of the lung. A review of reports in the literature would indicate that syphilis of the lung is more common than has been generally believed. The basis of the diagnosis has been the presence of an infiltration in the lung in a patient with a positive Wassermann and the subsequent clearance of the lesion by specific therapy. In our own experience we have looked carefully for pulmonary lesions in persons with syphilis in other parts of the body, and find that in the clinic as well as in autopsy material syphilis of the lung is very rare. The cases reported as syphilis often resemble our cases of fusospirochetal abscess. The sputum may be foul and the infiltration, as in fusospirochetal abscess, will resolve with arsenic therapy. In our experience fusospirochetal abscess may occur in a luetic person (Cases II and III), but that association is coincident. In one patient (Case II) who had a 4 plus Wassermann reaction the abscess showed little improvement with salvarsan therapy.

In the consideration of the treatment we will refer largely to our results from the use of neosalvarsan. It is, of course, recognized that some cases are amenable to surgery, others to bronchoscopy, but the type of case to which we have reference should be given a good trial with arsenicals before more radical procedures are instituted. The arsenicals have been employed extensively in the stubborn cases of Vincent's infection of the throat and about the gums. Prolonged cases of Vincent's angina and ulcerative gingivitis has been known to respond promptly to neosalvarsan. The use of salvarsan in the pulmonary infections has been less employed largely because of the failure to recognize the nature of the lesion. Our first experience was in

a girl, age eighteen, who in 1922 developed a pulmonary abscess following tonsillectomy with gas anesthesia. On the fourteenth day the expectoration became foul. The sputum examined at that time revealed many fusiform bacilli, cocci, and spirochetes. Fever continued, vomiting resulted from the stench, and for two weeks the patient became worse, lost weight, and was very discouraged. An *x*-ray disclosed a large cavity in the right upper lobe with considerable infiltration. She was given 0.45 gm. neosalvarsan and improvement was immediate. The expectoration became less; the temperature dropped. Two doses (0.6 gm.) were then administered at weekly intervals. An *x*-ray taken four weeks after the first revealed little evidence of an abscess. The patient ceased expectorating, gained weight, and has remained well to this date. It should be pointed out that such abscesses may heal spontaneously, but in this case as in several others, the response followed promptly after instituting treatment. It is in acute abscesses that the most gratifying results are obtained, for the more chronic the abscess becomes, the less response is seen with salvarsan. The acute lesions resolve often with no tendency to recurrence, but the chronic may improve with diminution in the expectoration, no fever for a variable interval, after which the patient returns with recurring symptoms. In abscess complicating bronchiectasis the response to salvarsan may be favorable. The sputum becomes odorless and less profuse, but the patient will return with recurrence, for the salvarsan can only influence the necrosis, but the bronchiectasis remains. There are instances in which the arsenicals apparently have little influence on the condition, and the failure is usually due to the pathology associated with the abscess. In abscess complicating diabetes no marked change was noted even when the treatment was combined with insulin. In some instances, as in Case VI, the process in the lung is rapidly progressive with the formation of multiple abscesses, and no treatment appears to influence the disease. If carcinoma or tuberculosis is associated the outcome may be dubious. However, in one instance in which we believe the abscess was associated with tuberculosis, improvement seemed to follow salvarsan therapy.

Case I.—J. R., white, male, age thirty-eight, entered the Cook County Hospital on September 17, 1926, complaining of cough, expectoration, pain in right chest, chills and sweats, and loss of weight over a period of two weeks. He was apparently well until two weeks ago except for an occasional cough. He developed chilly sensations with fever and began to cough. The cough soon became productive, noticeably becoming thicker and more profuse, particularly in the morning on rising. Pain was variable and worse with coughing. Perspiration was fre-

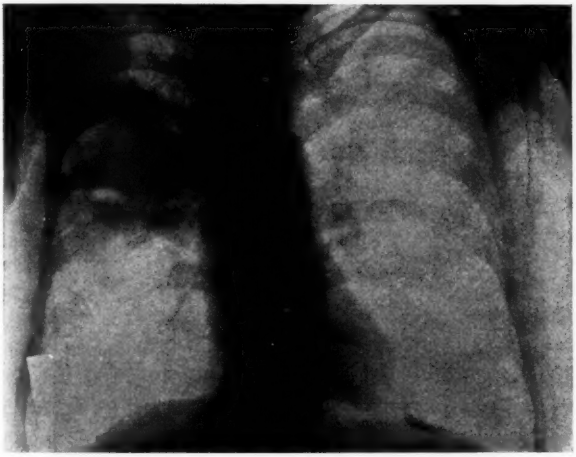

Fig. 9.—Case I. Lung abscess complicating tuberculosis before neosalvarsan therapy.

quent and profuse, accompanied by progressive weakness. He had no other symptoms of note, and the past, family and personal history disclosed no data of importance. Examination revealed a thin, flat chested person not acutely ill. There were many crowned and filled teeth, but gums were in good condition; the tonsils were not enlarged. In the chest there was impaired resonance over the right upper lobe with increased tactile and vocal fremitus. The breath sounds were bronchovesicular and numerous moist râles were heard. The remainder of the ex-

amination presented no abnormalities. The Wassermann test on the blood was negative. The temperature varied from 99° to 101° F. The sputum was profuse and foul smelling, and on standing formed typical three layers. In smears fusiform bacilli, spirochetes, and cocci were found. In cultures Streptococcus viridans, pneumococcus, and Staphylococcus albus were identified. The *x*-ray on September 20th reported a dense mottling of the right upper lobe with a rather sharp border along lower edge. Near the hilum was a uniform density about 3 cm. in diameter which was interpreted as a filled cavity of an abscess with tuberculous involvement of the right upper lobe. Several examinations did not reveal tubercle bacilli, but because of the apparent spontaneous development of the abscess a clinical diagnosis of

Fig. 10.—Case I. Symptoms of lung abscess have disappeared following neosalvarsan therapy, but infiltration in the upper lobe, interpreted as tuberculosis, remains.

tuberculosis of the lungs complicated by a pulmonary abscess was made. He was given 0.6 gm. of neosalvarsan intravenously at weekly intervals. After three doses the temperature became normal the patient felt improved, but expectoration was still profuse and foul. After the sixth dose the amount diminished markedly, the foul odor was absent, and only pneumococci were found in smears. At the seventh dose the *x*-ray reported evidence of small cavitations. After the tenth day only occasional cough and slight expectoration was noted. He had gained weight, was afebrile, felt stronger. The *x*-ray on December 20th reported a density suggesting a fibroid phthisis in the right upper lobe. On physical examination impaired resonance, but no râles were heard over this region.

It would seem that we were dealing with a patient afflicted with a slow form of tuberculosis which gave no symptoms except an occasional cough until fusospirochetal abscess was superimposed. The abscess apparently healed under therapy, but the old infiltration remained.

The following case presented a complicated picture in which the factors were not definitely clear. The involvement of the upper lobe without any distinct etiologic factor suggested tuberculosis as a basis. The patient had a four plus Wassermann, and neosalvarsan improved the general condition, but did not materially change the pulmonary pathology. Probably the subsequent course may throw further light on this particular case.

Case II.—H. M., fifty-four, white, entered the Research and Educational Hospital October 10, 1926 with complaints of cough for one year loss of weight and strength for three months. He stated that he had a continuous cough which became worse three months ago at which time he noticed that the expectoration became more productive. Pains in chest and sweats have been present for one month. The sputum was often blood tinged and noted to be foul only within the last month. The remainder of the history revealed no further positive information. Upon examination he appeared poorly nourished. Teeth were very carious. The chest presented dulness and râles in the right upper

lobe and axilla. The sputum was typical, forming the three layers, and in smears revealed fusiform bacilli, few spirochetes and cocci, no tubercle bacilli; the urine was normal; leukocyte count 13,800; Wassermann four plus positive. The *x*-ray report indicated a large cavity in the right upper lobe extending from the lower border of the first rib to the upper border of the third. The remainder of the apex showed some increase in density. During the following two weeks the fever rose usually in the afternoon from 99° to 102° F. He was then placed on weekly doses of 0.6 gm. of neosalvarsan for twelve doses. After three weeks the temperature seldom rose above 99.4° F., but the cough and profuse foul expectoration continued, although the patient was gaining weight and feeling generally improved. At the end of the course of injections the physical findings and *x*-ray still indicated considerable pathology in the right upper lobe. The abscess was slightly decreased in size and had a fluid level. The patient is now under observation in the surgical ward where other procedures will be tried.

In the next case the abscess developed without any outstanding etiologic factor, but observation later disclosed an old cavity or a pre-existing encysted empyema.

Case III.—N. W., colored, twenty-nine, chauffeur, entered the Cook County Hospital, October 18, 1926 complaining of pain in the left lower chest for twelve days and chils for five days. He was apparently well until twelve days ago, when he noticed a knife-like pain in the precordial region. This was inconstant for about seven days and then became more intense and constant. The pain was of a stabbing character, worse with deep breathing and coughing. Five days before entrance he had a chill lasting thirty minutes. Cough began only two days ago. In his past history there was disclosed a chancre six years ago and hemorrhoidectomy in 1917 without any complications. No other history could be elicited which would throw any light on his present trouble. Upon examination he appeared well nourished, not acutely ill. Temperature varied from 100° to 103° F., pulse 90 to 120. Leukocyte count was 12,000. Was-

sermann 2+ on the blood. Sputum culture revealed fusiform bacilli and pneumococci. The chest presented restricted motion on the left side, with diminished resonance in the right upper lobe and inconstant râles. On the 20th a friction-rub was noted over the left base. On the 21st the sputum was blood tinged and had a distinct odor. The *x*-ray reported on the 23d revealed a limited degree of density from the second rib to the fourth in-

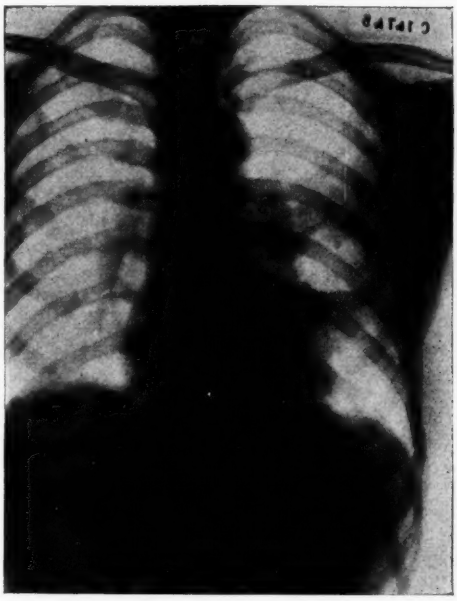

Fig. 11.—Case III. Abscess cavity with surrounding infiltration.

terspace, with a large cavity suggesting an abscess with surrounding infiltration. The sputum became more profuse and odor very foul, forming typical layers. In smears fusiform bacilli, spirochetes, and cocci, but no tubercle bacilli were demonstrated. On the 19th of November 0.6 gm. neosalvarsan was administered. Expectoration diminished markedly; the temperature occasionally rose to 100° F. On the 20th the same abscess cavity was noted

with less surrounding infiltration. The sputum became very scant, cough infrequent, temperature normal, and on the 29th the *x*-ray still showed the same annular shadow with no fluid level and no surrounding infiltration.

In the following patient the abscess complicated an old standing bronchitis and hypertropic emphysema, possible with bronchiectasis.

Fig. 12.—Case III. Annular shadow remaining after symptoms of abscess disappear.

Case IV.—T. A., forty-two, white, entered Cook County Hospital January 1, 1927 with complaints of cough, foul expectoration, chills and fever for three weeks, pain in the chest for two weeks. He had had a cough for many winters, but was otherwise well up to three weeks ago, when he began to expectorate foul sputum and have chills and fever. The cough be-

came worse and the sputum more profuse. No other factors could be elicited from the history. Upon examination he appeared ill, temperature 102° F., pulse 118. The teeth were poor; the breath very foul. The chest was emphysematous of the barrel-shaped variety. The upper lobe was impaired, while the remainder of the lung was hyperresonant. Over the right upper lobe a few râles were heard; the breath sounds were roughened and bronchovesicular. No other findings of importance were noted. The sputum was typical, forming layers, and contained numerous fusiform bacilli, few spirochetes, and many cocci and no tubercle bacilli. The leukocyte count was 12,000, polynuclears 85 per cent., small lymphocytes 13 per cent., 5 per cent. largely lymphocytes. The *x*-ray revealed an abscess cavity containing fluid in the right upper lobe from second rib to fourth rib. The temperature varied from 100° to 103° F. The patient is now under observation.

Case V.—Entered the Research and Educational Hospital of the University of Illinois on the service of Dr. Hedblom with complaints of fever, cough, foul expectoration for one month. The patient dates his trouble back to three years ago, when he had a similar attack in February, 1924, beginning as a cold and later developing what was diagnosed pneumonia. He recovered, and in February, 1926 again came down with a cough, expectoration of foul character, fever for about seven weeks. His present attack began with fever (103° F.) and profuse expectoration. At ten years of age he had had pertussis and at twelve rheumatism. Upon examination he appeared poorly nourished. dyspneic, with very foul breath. The teeth were poor; gums revealed pyorrhea. The chest presented increased tactile fremitus on the right side, with dulness in the right upper lobe to third interspace, and coarse bubbling râles in the same region. The heart revealed a diastolic murmur at the base. There was a capillary pulse and a blood-pressure of 112 systolic, 12 diastolic. A diagnosis was made of pulmonary abscess and aortic regurgitation. The blood showed a leukocytosis of 12,000, Wassermann reaction negative. The *x*-ray report indicated an area of

increased density in right upper lobe with lower border sharply outlined and the upper part irregular. Within this was an area of lessened density bounded below by a fluid level. The sputum formed typical three layers and contained numerous fusiform bacilli, moderate numbers of spirochetes, and cocci. In cultures the Streptococcus viridans appeared. The temperature varied from 98° to 101° F. On December 2d he had one rib resected and iodoform packing inserted as the first stage of the operation. On the 31st he received 0.3 gm. of neosalvarsan intravenously. On January 11th the packing was removed and the lung needled, but the abscess was not located. His cough became less marked, and sputum gradually reduced in quantity. His general condition improved, with gain in weight, and on February 6th he left the hospital. Lipiodol injection by Dr. Head at this time revealed no abscess cavity. It is probable that there was an old closed abscess cavity or an old encysted empyema that emptied at intervals.

The following case illustrated the rapidity with which the process may progress when the abscesses are multiple and therapy apparently has no influence on progress of the condition.

Case VI.—P. H., fifty-six, white, male, entered Cook County Hospital on October 4, 1926, with complaints of cough for four weeks, pain in chest one week, profuse expectoration of foul sputum for five days. These symptoms appeared to follow a cold of three weeks ago, which did not prevent him from working. Chilly sensations and sweats accompanied the other symptoms for the past ten days. Five days before entrance he suddenly began to bring up large quantities of foul sputum. History relative to past disease, family etc., revealed nothing of importance. No definite factor could be elicited. Upon examination the patient appeared a well-nourished adult male very acutely ill; temperature 101° F., pulse 120, respirations 30. There was marked fetor of the breath; the teeth were carious. The chest presented diminished expansion on the left side, impairment throughout left upper and upper portion of lower. The breath sounds were

harsh, bronchovesicular, and numerous coarse and crepitant râles were heard over the entire left side. The remainder of the examination disclosed no important findings.

The sputum was abundant, very foul, grayish green, and contained many fusiform bacilli, spirochetes, and cocci. The urine contained albumin and hyaline and granular casts. The Wassermann reaction on the blood was negative. The *x*-ray

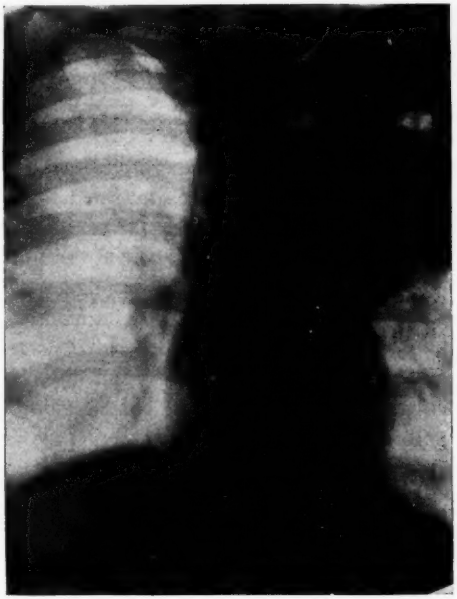

Fig. 13.—Case VI. Multiple abscesses of the lung.

reported extensive consolidation of the greater portion of the left upper, extending as an infiltration into upper half of left lower lobe, with the formation of intercommunicating cavities, one measuring 4 cm. in diameter. The extent of consolidation was interpreted as multiple abscesses complicating pneumonia. He remained very toxic with fever varying from 99° to 101.6° F., pulse 100 to 140; 0.3 gm. of neosalvarsan was ad-

ministered. Chills, sweats, and profuse expectoration continued, with no tendency to improvement, but rapid progress down hill. His condition became very poor, so that it seemed inadvisable to administer any more neosalvarsan, and he died on the twelfth day after entrance. Autopsy was refused.

CLINIC OF DRS. CHARLES A. ELLIOTT, WALTER H. NADLER, AND PAUL STARR

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LIVER FEEDING IN PERNICIOUS ANEMIA

THE favorable results reported in the past year with special diets in cases of pernicious anemia revive the hope that an effective treatment for this hitherto invariably fatal condition is available. Interest in the subject has been stimulated and light has been thrown on certain factors concerned with the pathogenesis of the disease.

The most valuable recent studies on the effect of diet on blood regeneration have been made by Whipple, Hooper, and Robscheit-Robbins. In carefully controlled experiments on dogs they showed that both hemoglobin regeneration and bile-pigment excretion are influenced by modifications of diet. Since it is recognized that bile-pigment is probably the waste product of hemoglobin metabolism, and that pigment metabolism is an important process constantly going on in the body, studies of these substances are of particular interest in pernicious anemia. Whipple and his co-workers found that bile-pigment excretion is increased on a carbohydrate and decreased on a protein diet, while hemoglobin regeneration is slow on a carbohydrate and rapid on a protein, especially on a liver, diet. Speculation as to what may be present in liver or kidneys as a food that will stimulate blood formation is of exceeding interest. They are rich in nuclein, and contain a high quality as compared with a relatively low quantity of protein. It has been suggested by Whipple that a deficiency of erythrocyte reticulum material

may be present in pernicious anemia. The hypothesis that pernicious anemia may be a disease due to a diet deficient in substances required for the building of the stroma of the red cells is supported by the presence of very numerous, irregular "tag-ends" of red cells apparently well filled with pigment. It has been suggested by Koessler that pernicious anemia is a vitamin deficiency disease. The liver has a vitamin content probably higher than that of any other organ. The experimental production of severe anemia in animals maintained on a deficiency diet and its correction by the administration of vitamins lends support to this theory. The high iron content of liver is probably not a factor in the remissions observed on liver feeding, since there is no iron deficit in pernicious anemia.

Clinical application of Whipple's diet was made by Gibson and Howard, who in 1923 advocated the use in pernicious anemia of an iron-rich and vitamin-adequate diet. Minot and Murphy in 1926 reported remissions by the use of a special diet in 45 cases of pernicious anemia observed essentially in sequence. The remissions had been maintained in all except 3 patients who discontinued the diet during the six weeks to two years under observation. These patients were given food rich in complete proteins and iron, particularly liver (*120–240 gm. cooked calf's or beef liver daily*), an abundance of fruits and fresh vegetables, and a relatively low fat intake. All showed a prompt and distinct remission of their anemia and a general improvement, save only in pronounced disorders due to spinal cord degeneration. Koessler, Maurer, and Loughlin advocate the following vitamin theory of pernicious and other severe anemias: That the blood and gastro-intestinal changes may be due to chronic vitamin A underfeeding, the nervous symptoms to vitamin B deficiency or absence, and the hemorrhagic tendencies to partial or complete lack of vitamin C. They recommend a diet containing *liver or other high nucleoprotein food* (*100 gm. daily*), fresh fruit and vegetables, a relatively high fat content in the form of butter, cream, milk, and egg-yolk, and the addition of cod-liver oil (5 c.c. four times daily), yeast tablets, and dilute hydrochloric acid (30 drops before, during, and after each meal).

CASE REPORTS

Our experience with liver feeding in pernicious anemia will be illustrated in detail by the presentation of a patient who has experienced a definite remission. The results in other cases will be summarized in a table. In addition, a patient with severe anemia, but not of a typical "pernicious" type, will be shown as one who has not responded to dietary measures. Our plan of treatment of pernicious anemia was changed in May, 1926 to include the addition of liver; ¼ to ½ pound, cooked or raw, has been given daily either with meals or by stomach-tube. Other measures have been continued as heretofore, without fixed rule, *i. e.*, dilute hydrochloric acid, cod-liver oil, Quartz lamp exposures, iron, and arsenic.

Case of Pernicious Anemia with Remission on Liver Feeding.—Mr. S. J., a cattle-buyer, forty-nine years of age, whose work was largely out-of-doors, was under observation in Wesley Memorial Hospital from March 26, 1926 to January 9, 1927. On admission he complained of weakness and of tingling in his hands and feet. These had been present for two years, gradually increasing in spite of some periods of improvement. He had lost but little weight. For nine months he had had some difficulty in picking up objects. His sense of touch seemed less acute; he occasionally dropped objects he was holding. The only gastro-intestinal symptoms were lack of appetite and constipation; sore tongue was not mentioned. Slight dyspnea, palpitation, vertigo, and swelling of the feet had been noted. There was no renal disturbance. He had had no serious illnesses. His mother died of carcinoma; an aunt, of tuberculosis.

Physical examination found a well-nourished white man with slightly lemon-tinted skin. The mucous membranes were very pale, the scleræ were clear. The thyroid was palpable; the lungs were clear. The heart was normal in size, with no murmurs; blood-pressure 120/70. The spleen was not palpable, but had been felt in the fall of 1925. The liver was not enlarged. The genitalia were normal. No abnormal reflexes; no gross loss of joint sense; vibratory sense was maintained over the tibiæ.

There was no spasticity. The remaining teeth showed slight periapical infection; nose, throat, and ears negative.

The laboratory data on admission included:

R. B. C., 1,020,000.

W. B. C., 4400.

Hb., 24 per cent.

Color index, 1+.

Smear: Marked poikilocytosis.

Differential: Poly., 58 per cent.; lymph., 40 per cent.; baso., 2 per cent.

Wassermann negative.

Stool well formed, benzidine for blood negative; microscopic negative.

Van den Bergh estimation on August 31st normal (0.49 mg. of bilirubin per 100 c.c. of serum).

x-Ray of G. I. tract and chest (9/28/25) negative.

Fractional test-meal (3/20/26): No free acid; nine single stomach specimens taken by tube on different days during September and October of 1926 were negative for free acid.

Progress and Treatment.—The nine and one-half months' stay in the hospital may be divided into two periods, the first, of five months, during which he had the standard treatment for pernicious anemia with the usual gradual failure; the second, of four and one-half months, during which he received liver by stomach-tube daily, at the end of which period he had recovered except for cord degeneration and muscular weakness (Fig. 14).

In the first period he received the following treatment:

1. Dilute hydrochloric acid 50, well diluted with each meal.
2. Cod-liver oil, 4 ounces daily.
3. Iron citrate, amp. 1, every second day.
4. Ultraviolet light exposure daily.
5. Fruit, meat, and vegetable diet.
6. In August his mouth and tongue became so sore that he required a mouth spray of 5 per cent. cocain before meals; powdered opium was occasionally used for diarrhea.

7. Eight blood transfusions were given, three of 500 c.c., and five of 300 c.c., of citrated blood from May 10th to August 28th.

In August, at the close of the above régime, he was confined to bed because of extreme inanition. The muscles of his legs, especially, were greatly wasted. His tongue for two months had been growing more and more atrophic, pale, shiny, and painful. His red blood-count fell to less than 1,000,000 in April, rose to 2,000,000 during May, June, and July, but declined again to

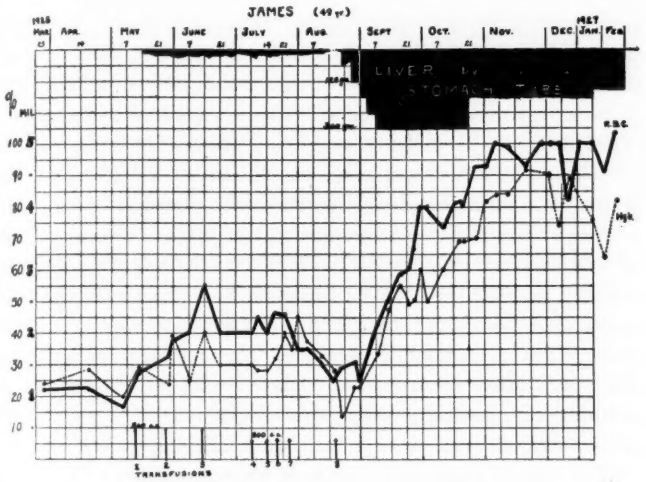

Fig. 14.—Liver feeding in pernicious anemia.

1,250,000 in August, the hemoglobin ranging from 20 to 30 per cent. at best. During the latter part of May, June, and July it was attempted to add liver to his diet, but he had always had a marked distaste for liver, and refused to do more than chew a bit of it, after which he spat it out. From August 25th to 30th he was persuaded to try to eat about 150 grams of beef liver a day, but this was too fatiguing, especially because of his sore tongue.

From August 31st to October 21st he received an average of

300 grams of puréed, parboiled beef liver by stomach-tube daily. Since then, *i. e.*, for five months, he has taken about 150 grams a day by the same method. The previous medication, including the hydrochloric acid, has been continued as before. The clinical transformation began within two weeks, and has resulted in a gain of more than 25 pounds. Regeneration of the tongue began early and was complete in ten days. This constituted one of the most striking changes and was complete before any marked effect on the blood was evident. The blood-count rose rapidly from 1,200,000 on August 31st to 4,000,000 on September 29th (hemoglobin 60 per cent.), and then gradually to 5,100,000 (hemoglobin 83 per cent.) on November 4th, and to date (after six months) is over 5,000,000 (hemoglobin 85 per cent.). The blood-smear rapidly approached normal until in November only slight traces of irregularity could be found. The reticulate cell percentage became 5 per cent. on September 13th and reached 10 per cent. on September 25th, and since then has continued at 3 or 4 per cent. By October 4th he was up in a wheel-chair; on October 9th he stood upon his feet for the first time in many months, and by the 15th he walked across the room. He now walks with the aid of a cane and can climb stairs without assistance. Muscular growth paralleled this recovery of function. His gait is ataxic; coarse joint control is still lacking owing to a combination of weakness and cord degeneration. It is difficult to demonstrate sensory or reflex abnormality in the legs.

Ten Other Cases of Pernicious Anemia on Liver Feedings.—All showed prompt evidence of blood regeneration. Four were observed to reach a red count of 4,000,000 within five to seven weeks. Five have obtained a complete remission; in one of these cord changes have progressed, although his general health is good. Three patients, observed for three to six weeks, have not yet reached a 4,000,000 count. Two elderly patients with extreme inanition showed prompt improvement, but developed bronchopneumonia and died. It is possible that the necessity of feeding liver to these patients by stomach-tube played a part in the development of the purulent bronchitis and bronchopneumonia observed at autopsy (Chart 1).

CHART 1

	Red blood-cells before liver.	Progress.	Present count 3/10/27.	Duration of diet.
1. Mr. L.	2,250,000	.....	4,700,000	10 months
2. Dr. M.	2,100,000	6 wks. 4,000,000	4,900,000	7 "
3. Mrs. W.	1,500,000	7 wks. 4,000,000	4,500,000	4 "
4. Mrs. K.	1,000,000	6 wks. 4,000,000	.....	10 weeks
5. Mr. H.	3,000,000	5 wks. 4,000,000	4,600,000	6 "
6. Mr. G.	1,000,000	4 wks. 3,000,000	3,000,000	4 "
7. Mr. G.	900,000	3 wks. 2,000,000	2,000,000	3 "
8. Mr. F.	900,000	5 wks. 3,200,000	3,200,000	6 "
9. Miss H.	1,200,000	2 wks. 2,300,000	Died, bronchopneumonia	3 "
10. Mrs. McK.	1,200,000	3 wks. 2,500,000	Died, bronchopneumonia	4 "

Case of Severe Anemia Without Remission on Liver Feeding.—Mrs. A. H., age thirty-two, the mother of 3 healthy boys, was admitted to Wesley Memorial Hospital on September 11, 1926. She felt perfectly well until eight years ago (1918), when she had a severe postpartem hemorrhage, from which she never fully recovered, in that she retained a degree of anemia. In 1919, after a streptococcus sore throat, she had a severe uterine hemorrhage for one month. In 1921 she had bleeding from hemorrhoids for a year. She was fairly well until 1925, when her count was 1,600,000. In March, 1926 she had an acute respiratory infection. Two infected teeth were extracted with considerable bleeding. Three blood transfusions were given after this to relieve weakness and tachycardia.

Physical Examination.—A fat young woman with definitely icteric scleræ and yellowish skin. Nose, throat, teeth, and tongue normal. No goiter. Lungs clear; heart enlarged, with systolic murmurs. Abdomen negative; liver and spleen not felt. Pelvis negative; uterus normal in size and shape, slight prolapse. Proctoscopic negative. Reflexes and sensation normal; vibratory sense is normal. There is no atrophy or spasm of the leg muscles.

Laboratory data:

R. B. C., 360,000 (on admission); 520,000 (later).

W. B. C., 1900.

Hb., 13 per cent.
Color index, 1.85.
Smear: Poikilocytosis is not marked; anisocytosis considerable; cells are well filled; many large pale red cells; reticulated red cells 8 per cent.; platelets appear frequently in smear.
Differential: Lymph., 90 per cent.; polys., 8 per cent.; eosin, 2 per cent.
Fragility (dilution resistance) red cell test, normal (0.46-0.38).
Bleeding time two to three minutes.
Clotting time two and a half minutes.
Van den Bergh (April, 1925) three times normal.
Wassermann negative.
Stomach fractional test, Free HCl 20, combined 20.
G. I. *x*-ray negative.
Sinus ray: Slight clouding of left submaxillary.
Stool: Soft, brown, no blood, no parasites.
Urine: Alb. trace; sugar 0; Sed., no R. B. C.
Progress and treatment:

1. Dilute HCl, 50 t. i. d. with meals.
2. Diet to contain orange juice, tomato juice, vegetables, and meat.
3. Liver, 200 grams, parboiled, daily; recently 100 grams raw, 100 grams parboiled.
4. Cod-liver oil, 1 ounce, t. i. d.
5. Iron arsenite, 1 grain every other day; recently Blaud's pills iii, t. i. d., by mouth in addition.
6. Ultraviolet light exposure (omitted after few weeks).

During the first three weeks in September the count rose gradually to 1,400,000 (hemoglobin 30 per cent.). During December it ranged about 2,500,000 (hemoglobin 42 per cent.). Most of the course was afebrile, but in the first two weeks of November a steady fever of unknown origin began, running to 101° F., with a rapid fall of the count to 390,000. At this point a transfusion was done with marked reaction, but subsequent cessation of fever, and the count returned to 1,200,000, and later

rose. A second transfusion was given in December. There was a slight febrile reaction and marked hemoglobinuria. Following this eleven slightly infected teeth were pulled without bleeding. The coagulation time was then one and a half minutes. During January the count gradually declined to 1,900,000. On the theory that a chronic sepsis existed, a non-specific protein shock with 10,000,000 typhoid vaccine intravenously was given on January 31st. This was followed by a high temperature and by marked hemoglobinuria for three days and a fall of the red count from 1,900,000 to 1,300,000, but especially by a fall of the hemoglobin from 30 to 17 per cent. Since then, during February, the red count continues at from 1,000,000 to 1,600,000; the hemoglobin from 18 to 27 per cent. Menstruation has occurred several times, but is brief and scant. There have been several slight respiratory infections. The general condition of the patient is remarkably good in relation to the severity of the anemia. Nutrition is excellent, appetite and bowels are normal. Usually there is no fever; the pulse is about 90. She is confined to bed.

The diagnosis in this case is difficult. The condition is probably one of insufficient blood formation, *i. e.*, aplastic anemia with occasional purpuric features according to the history of hemorrhages (although the last platelet estimation was 150,000), associated with constant and occasionally great blood destruction as evidenced by icterus and hemoglobinuria. Blood destruction is evidently not due to fragility of the red cells, since their resistance is normal. That this is apparently not a case of pernicious anemia (of Addison's type) is indicated by the presence of free acid in the stomach, the lack of tongue atrophy, and the, absence of cord changes. Liver administration has been without apparent benefit.

CONCLUSION

The application of liver feeding to cases of pernicious anemia suggested by recent experimental work has added to the therapeutic armamentarium of this disease an agent that gives results not approached by any other measures. The clinical fact seems established that liver added in sufficient quantity to the diet of typical pernicious anemia patients will produce a remission.

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A CASE OF ACUTE MYELOBLASTIC LEUKEMIA WITH ALEUKEMIC COURSE, WITH AUTOPSY FINDINGS

THE patient whom I wish to present today is a German, forty-four years of age, and by occupation a trimmer of rubber tires, working in a tire factory. I first saw the patient in private practice, being called into consultation with the family physician because of the symptoms which I will enumerate shortly. A very cursory examination showed that the case was one of unusual interest as well as severity, so that as his means were limited I suggested his coming to the hospital for a careful study of the case.

He entered the hospital on May 14, 1926, with the following history:

Present Complaint.—1, Nosebleed; 2, bleeding from the gums; 3, black stools; 4, weakness; 5, severe headaches; 6, vomiting of blood; 7, dizziness and failing vision; 8, bluish-black spots over the entire body. The duration of these symptoms had been about four weeks and they were becoming progressively and rather rapidly worse.

Onset and Course.—The patient stated about four weeks ago after returning home from work his nose began to bleed without any apparent cause. This continued for four or five hours and stopped spontaneously. At nine the following morning bleeding from the nose reappeared and attained such a degree of severity that a physician had to be called, who packed the nose. It was repacked several different times before the bleeding was controlled. He thinks he lost a great deal of blood.

From that time to the present the patient has been confined to bed most of the time owing to the progressive weakness. Bleeding from the nose has recurred at irregular intervals. About two weeks before his admission the patient noticed numerous bluish-black spots on the legs and arms. He called these to the attention of his physician. The spots gradually disappeared. A week ago the gums began to bleed, but they did not feel particularly tender. At no time has the mouth or tongue been sore, and at no time had his attending physician noticed anything unusual about the buccal cavity. Following the bleeding from the mouth the stools were very dark and dark stools are still present. During the past week the patient has also vomited three times on three successive days. In each case the vomiting followed the intake of food. The first time the vomitus was bright red and the second and third times of a chocolate brown. He insists that the appetite was good until the onset of the vomiting, since which time it had been very poor. Dizziness and frontal headache have been present since the onset four weeks ago. During the past two weeks the vision has been steadily failing, the far vision being especially impaired.

General Symptoms.—Nervous symptoms are entirely absent with the exception of the headache and dizziness already described.

Respiratory.—He has a little cough at times productive of mucus, but there is no history of hemoptysis. He has had some shortness of breath since the beginning.

Cardiovascular.—Palpitation is the only symptom worthy of mention in this category.

Gastro-intestinal.—The vomiting of blood, especially in the absence of pain or gastric distress, is striking. The dark stools I may say were actually due to blood, and blood is present at this time in considerable quantity. The bleeding gums have already been referred to.

Failing vision is the only symptom on the part of the special senses.

Genito-urinary symptoms are absent, and in particular there has been no hematuria.

Musculocutaneous.—He does not think he has lost any weight, but the muscular weakness is extreme. The bluish-black spots on the skin have already been referred to as hemorrhages.

Past History.—Medical: Pneumonia at sixteen, typhoid and malaria at twenty-eight. Surgical and venereal abnormalities denied.

Family History.—Mother is living and well at seventy-seven; father dead of an accident at sixty-six; one sister and two brothers living and well. He has had 5 children by a former marriage, all living and well. There is no history of tuberculosis, carcinoma, or bleeders in any of the family in this or former generations.

Habits.—Alcohol to excess from nineteen to twenty-nine years of age.

Physical Examination.—The essential points of this as determined upon his entrance to the hospital are as follows: A fairly well-developed and nourished white male of forty or forty-five years of age. Evidently acutely ill. There is dry blood about the nostrils and teeth and actual bleeding from the left nostril. The breath is very foul.

Regional Examination.—Hair and scalp substantially negative. Ears: Hearing is good; no mastoid tenderness; no tophi or discharge. Eyes: Vision is greatly impaired; conjunctiva is very pale, the sclera bluish white, the cornea clear, pupils reacting to light and accommodation. Extra-ocular movements normal. Nose: Dry blood about both nostrils and small amount of fresh hemorrhage from the right side. Both nares are completely obstructed and the patient is bleeding through the mouth. Further examination of the nose deemed inadvisable because of the bleeding. Mouth: Lips are dry and coated with dry blood. The mucous membranes are very pale. The tongue protrudes in the midline and there is a heavy brown coating over the posterior part. There is no evidence of swelling or inflammation. The gums show evidence of old and recent hemorrhage. The teeth are partly covered with dry blood. They are not spongy, but bleed on very slight trauma. Blood can be seen dropping from the posterior pharynx.

Neck: Movements are free; anterior cervical glands just palpable; thyroid not palpable.

Chest: Symmetrical, fairly well-developed, with fair and equal expansion. There is no lag. Litten's sign present.

Lungs: Tactile fremitus normal, with normal resonance throughout. There are many moist râles, medium-sized, scattered over both lungs anteriorly and posteriorly.

Heart: Apex-beat is visible in the fifth interspace in the midclavicular line; the borders are normal; the tones are regular and rather weak; a soft, blowing murmur heard at the apex not transmitted; the second pulmonic sound is reduplicated.

Abdomen: This is slightly distended and many small purpuric spots are to be seen over the lower chest and abdomen. The liver is palpable three fingers below the costal border. It is regular, firm, not tender, slightly rounded. The spleen is palpable three fingers below the costal border, somewhat softer than the liver. The notch is easily palpated. The abdomen is tympanitic throughout. No fluid wave obtained. The inguinal lymph-glands are just palpable.

Extremities: Normal except that many purpuric spots are scattered over the lower ones. The reflexes are normal.

In the discussion of the diagnosis I should like to consider the case as I first saw it in consultation, at which time no laboratory work had been done. I think you will all agree with me that one of the first things to be thought of was benzol poisoning inasmuch as the man worked around tires. The most careful questioning not only of the patient but of fellow workmen showed that he had very little if anything to do with benzol. His actual job was to trim the tires mechanically. At best, this diagnosis could hardly be maintained in view of the evident enlargement of the spleen and liver, since these are absent in benzol poisoning, at least absent to a degree clinically recognizable. In view of the very strongly pronounced hemorrhagic tendency, our thoughts would turn at once to those diseases associated with profuse hemorrhages and an enlarged spleen. The patient was running a temperature around 101° or 102° F., so that some thought had to be given to the hemorrhagic forms of the acute infections.

However, these were easily excluded, since such infections as smallpox, meningitis, ulcerative endocarditis, were hardly consistent with the history given. The two things which seemed most probable were purpura hæmorrhagica and acute leukemia. In both of these diseases fever is present, the spleen generally enlarged, hemorrhages from various organs and into the skin quite common, great weakness and prostration may occur. As

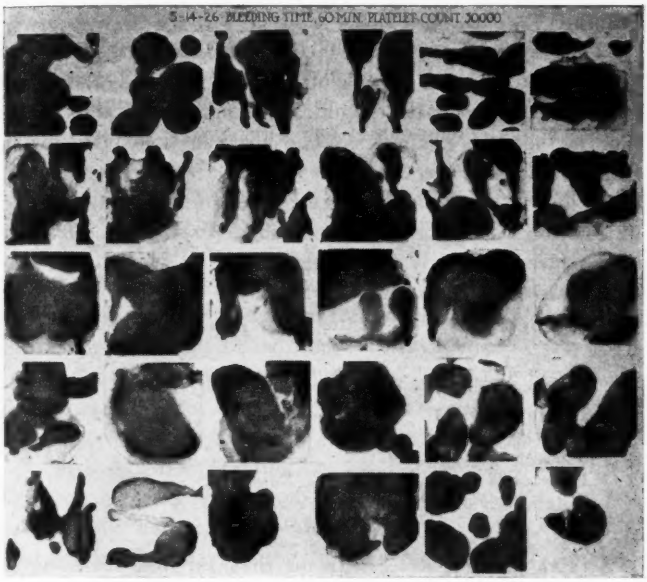

Fig. 15.—Aleukemic myelosis bleeding time on admission.

between the two, we find that acute leukemia generally has, at least after a month's duration, marked inflammatory conditions about the tongue and buccal cavity. This was absolutely not the case in our patient. In the lymphatic types of leukemia the peripheral glands are generally enlarged, which was not the case either, since the glands were just barely palpable. I very frankly admitted my inability to make a diagnosis in the absence of a careful study of the blood. It may be that some of you are think-

ing of the possibility of scurvy, and that, too, had to be considered. Inquiry, however, showed the patient had been living on a fairly good diet and, indeed, the whole course of the disease was much too stormy to admit of the diagnosis of scurvy, to say nothing about the enlarged spleen, which I have never seen in a case of scurvy.

Needless to say, a careful study of the blood was undertaken as soon as the patient came into the hospital, with the following results:

The erythrocytes were 900,000, the leukocytes 4350; the platelets 30,000, the hemoglobin taken by the Newcomber method 22 per cent.; the coagulation time taken by the capillary tube method three minutes. The bleeding time taken by the Duke method *was over sixty-nine minutes*. A very tiny puncture in the lobe of the ear bled for sixty-nine minutes freely, at which time cotton was applied and efforts made to check it, but it continued bleeding slowly for another hour, at which time horse-serum was used to stop the bleeding.

The differential count of the leukocytes was most interesting:

	Per cent.
Polymorphonuclears	59
Small lymphocytes	20
Large lymphocytes	15
Transitionals	5
Eosinophils	1
Unidentified	8

I think you will agree with me that, far from lessening our difficulties, these blood findings rather tended to increase them. To begin with, it is practically impossible to make the diagnosis of acute leukemia during an aleukemic period (which, of course, we all know to exist), unless the differential count is very decisive. A great deal of patient and laborious study was given to these blood-smears and the cells noted as unidentified were really almost impossible to classify. I may say, however, that we have since come to regard some of them as very atypical myeloblasts and myelocytes. It is hardly necessary to say that very frequent, almost daily examinations of the blood were made and

studied by many of the staff especially versed in hematology. The general impression of these cells was that they were immature, but, of course, that means very little except that there is a very active bone-marrow, and that some of the newly formed cells are being swept into the circulation. Careful search in almost any severe hemorrhagic condition associated with regeneration will show a few such cells in the peripheral circulation. In other words, we might very well have a few such cells in a severe purpura hæmorrhagica, as I have seen more than once.

While of no great diagnostic importance, several other tests were made, especially the prothrombin test (Howell), and this was found to be fourteen minutes. A fragility test was undertaken by the ordinary technic, and it was found that hemolysis began at 0.38 per cent., and was complete at 0.36 per cent. It is very important that a normal control be made in this test, and our normal began to hemolyze at 0.4 per cent. and was complete at 0.36 per cent. The coagulation time also taken by Howell's method, was thirty minutes. The clot did not contract for many hours, and late at night was transferred to the refrigerator, and the test-tube on the following morning contained three layers, a top layer of clear straw-colored serum, a middle layer of solid white elastic character, and a lower layer of dark red fluid containing all the blood-cells.

Immediately after entering the hospital 100 c.c. of blood was given intramuscularly, and on the following morning 540 c.c. intravenously, and the same afternoon 600 c.c. intravenously. The following day the erythrocyte count was 1,790,000; platelets, 70,000; hemoglobin, 37 per cent. The interesting feature, as showing the value of transfusion in these hemorrhagic conditions, is that the bleeding time was reduced to *four and a half minutes*. This bleeding time, as you know, is, in a large measure at least, a function of the platelets. When the platelets get down much below 100,000 hemorrhages are prone to occur, and when they get down below 50,000 hemorrhagic phenomena are the rule and become increasingly severe with the diminution in the number of the platelets. The second morning the erythrocytes were 1,650,000; the leukocytes, 5600; platelets, 80,000; hemoglobin,

27 per cent. During all this time bleeding was undoubtedly going on from the intestines, since the stools contained considerable quantities of blood.

On the 19th another transfusion of 500 c.c. was made. Bleeding time four minutes; platelet count remained 80,000. On the

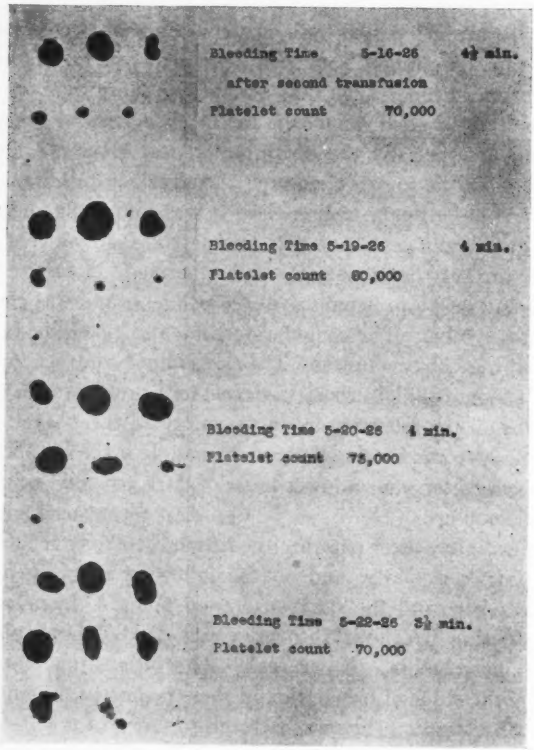

Fig. 16.—Aleukemic myelosis bleeding times and platelet counts.

20th the erythrocytes were 2,440,000; the leukocytes, 4850; platelets, 75,000; hemoglobin, 42 per cent.; bleeding time, four minutes. The differential count showed polymorphonuclears 55 per cent.; small lymphocytes, 18 per cent.; large lymphocytes, 15 per cent.; transitionals, 2 per cent.; eosinophils, 2 per cent.; un-

identified cells, probably myeloblasts and myelocytes, 8 per cent. These differential counts were made with the utmost care by Dr. Birch. The increase in the number of large lymphocytes is noticeable.

I forgot to mention that as a matter of course blood-cultures were taken repeatedly and showed no growth either in aërobic or anaërobic cultures. The tourniquet test was made on several occasions, with the following results: Petechial hemorrhages would appear at the end of one minute and many of them would occur at the end of three minutes. This is, of course, nothing more than the measure of the capillary resistance and is generally looked upon as semipathognomonic of purpura hæmorrhagica.

The course of the disease in this patient was steadily downward. The stools always contained blood, and when vomiting occurred it also showed blood. On the 26th in spite of more transfusions the red blood-count was 1,410,000; white count, 10,500; hemoglobin, 25 per cent.; platelets, 55,000. The patient was bleeding all day. We asked our surgical colleagues to consult with us and to consider the advisability of doing a splenectomy. Our train of thought was about as follows: With a diagnosis extremely difficult and with the patient not showing any signs of a remission, it was quite evident that a fatal end was near. He had, therefore, everything to gain and nothing to lose by such an operation. If the case were really a genuine thrombocytopenic purpura, such an operation if successful, is often life saving. Whereas, if the case were an acute leukemia, it made very little difference in the outcome.

Three Days Later.—On the 26th and 27th two additional transfusions of 500 c.c. each were made, and just before the patient was placed on the operating table, on the morning of the 27th, the erythrocyte count was 1,990,000; white count, 8500; platelets, 70,000; hemoglobin, 39 per cent. Patient was transferred to the operating room and an attempt made to remove the spleen which had been growing steadily larger, but without avail. The patient died before the operation had fairly commenced. An autopsy was obtained and the following is an abstract of the postmortem record:

Acute aleukemic myelogenous leukemia; great enlargement of the spleen, which weighed 1175 grams. Its size was 20 x 12 x 6 cm. Myeloid metaplasia of the splenic pulp. Very immature blood-cells predominated. Myeloid metaplasia of the lymph-glands about the pancreas and the abdominal aorta. Aberrant spleen about the tail of the pancreas which showed the same changes as the main organ. Moderate enlargement of the liver (2090 grams), with accumulation of myeloblasts, normoblasts and giant-cells in the portal capillaries. Metaplasia of the marrow of the long bones, in which are found a great many myeloblasts and groups of erythroblasts. Groups of myeloid cells in the capillaries of the cortex of the adrenal body and of the kidney.

Beginning fatty degeneration of the myocardium. Diffuse colloid goiter. Recent hemorrhages in the skin of both arms, right upper abdomen, and right thigh. Hemorrhages in the mucosa of the fundus of the stomach. Ascites, small amount. Recent operative wound in the left hypogastrium.

Discussion.—In the light of the very definite pathologic findings, and especially in view of the very careful histologic examination of the organs, there can scarcely be a doubt that our case was, in reality, an acute myeloblastic leukemia. The following points are, however, worthy of a little more extended consideration: The aleukemic condition of the blood is well known to occur occasionally in many cases, but rarely ever lasts any length of time. A small number of cases have been recorded in which the aleukemic condition persisted throughout the disease and it seems quite probable that that was the case with our patient. In any event, he was not only aleukemic, but leukopenic during most of his stay in the hospital, and at no time was even a slight leukocytosis noticed. We debated the question of the atypical cells in the blood at great length. It was exceedingly difficult to classify them, and it is probable, now that we know the histology of the organs, that they were atypical myeloblasts. Would we have been justified in diagnosing the case positively on the basis of this number of myeloblasts of such a character? I think there might well be a difference of opinion

on this subject, since every clinician of experience must have seen severe anemias with a modest number of myelocytes and even myeloblasts in the peripheral circulation. The greatly diminished number of platelets is, of course, characteristic of the genuine thrombocytopenic purpura. On the other hand, in many conditions associated with severe bleeding the platelets may be low and acute leukemia is no exception to this rule. The tourniquet test, upon which some authors lay considerable stress as being diagnostic of purpura, was positive in this patient. It has never seemed to me that this test was reliable either in principle or practice, since at best it cannot indicate anything more than badly damaged capillaries from any cause.

One of the most interesting features of this patient is the absence of all inflammatory signs about the mouth. I have had the good fortune (or perhaps I should say misfortune) to see a considerable number of cases of acute leukemia, and I think this is the very first in which I have missed severe inflammatory changes about the mouth or the tonsils or tongue. In one instance a small blister at the tip of the tongue rapidly proceeding to gangrene of over half of that organ was the first symptom of which the patient complained. In still another the gangrenous tonsillitis brought the patient into the hospital. A third showed gangrene of the cheek which was diagnosed as noma, and a blood examination more or less accidentally made was the first intimation of the leukemic condition. I might instance several other similar cases.

To sum up the entire case we have a most unusual and most difficult acute myeloblastic leukemia in which the difficulties of diagnosis were extraordinarily great, and in which, despite careful study by a group of men well versed in hematology, the diagnosis could not be positively made.

A CASE OF HEMOPHILIA, WITH GENEALOGICAL CHART

THE patient for our clinic today is a young Jewish boy sixteen years of age, who came into the hospital some time ago with the following history:

Present Complaint.—Bloody urine; pain on the left side of the abdomen; extravasation of the blood on the inner aspect of the right arm; multiple purpuric spots over the lower extremities ranging in size from a bean to that of a half-dollar. These have all come on within the past three days. In addition to this he complains of nocturia, five to six times a night, with dysuria when he passes clots of blood, which happens quite frequently. He complains of great pain in the knees when flexion is attempted, and of limitation of motion of the elbows, knees, ankles, and left hip.

Onset and Course.—The lad states that he was feeling in his usual good health until three days ago when he suddenly urinated considerable quantities of bloody urine. He fainted at the sight of the blood, although he had no pain. Twice during the following night he passed blood. The next day he went to a hospital dispensary, where they administered thromboplastin. The following night (Friday) he still continued to pass blood and Saturday he was admitted to this hospital.

Similar Attacks.—Last September (1925) the patient had a similar attack. He then had marked hematuria, and this was accompanied by abdominal pain. The subcutaneous hemorrhages, he states, were also present in that attack and he thinks they are caused by trivial traumata. He states that he was not a cripple at birth, but that his left hip and knee became deformed after he started to school. He has worn many casts and braces.

Past History.—Patient was a full-term normal baby. Normal delivery, weight $7\frac{1}{2}$ pounds. When three weeks old he was circumcised and bled profusely and was taken to a hospital for treatment. At this time he bled for two weeks, and the patient

states that it was stopped only after the use of thromboplastin. Following the circumcision he had no further trouble for six months, and at this age the mother states that he was a fat, handsome baby. At this time he had a fall and the following day his entire face was black and blue, the extravasation of blood being out of all proportion to the severity of the fall. At two years of age he fell and cut his lip slightly and from this cut he bled pretty constantly for two weeks. Since this time he has bled on many occasions very severely and always after what would be to a normal child a trivial injury. He has had spontaneous hematuria to a marked extent on a number of occasions in addition to those mentioned. Epistaxis has occurred occasionally, but never to an alarming extent.

Medical History.—Measles, mumps, and chickenpox are the only diseases our patient has had. His surgical history is entirely negative excepting, for circumcision.

I think perhaps that the most useful and interesting way of discussing this patient is to see what can be made out of the case from this much of the history alone. A perusal of the points I have just made indicates two things: first, that this youngster bleeds on the smallest provocation for very long periods of time; second, that the hemorrhages are spontaneous as well as traumatic, and most important of all, that they have existed since his earliest infancy. When we ask ourselves what conditions are associated with such a history the answer is very few. Toxic conditions would be almost certainly eliminated by the age at which the hemorrhages developed. Leukemia, of which we have just had such a striking case, does not give this kind of history. The same thing might be said of aplastic anemia, which occurs in childhood. Purpura hemorrhagica, or as we now prefer to call it thrombocytopenia, rarely ever develops at such an early age, seldom produces such prolonged hemorrhages, and while remissions and exacerbations occur, I have never known a purpuric patient to have a history in any way even approximating the present. As a matter of fact, the history is so characteristic that it is difficult to avoid making what might be called a snap diagnosis of the great classical hemorrhagic condition, namely,

hemophilia, the true bleeder's disease. While not perhaps entirely apropos of this case, I should like to say that there are few words more badly abused by physicians than the term "bleeder." It is a common experience to have patients, both women and men, consult one, and in the course of the history they state that they are bleeders. Not only this, but in a goodly number of cases it is extremely difficult to disabuse their minds of this idea. They have been told by several physicians who know them very well that they are bleeders, and bleeders they are determined to be. Now it takes a good deal more than one or two copious hemorrhages from any source to justify a diagnosis of a true bleeder. Not only must the hemorrhages occur spontaneously or from trifling causes, but they must occur repeatedly and most important of all, they must be definitely hereditary. A few years ago we would have added another point and said that they must occur in the male. It still is true that the overwhelming majority of bleeders occur in male patients, but I think the best opinion is that, although very rare, it may occur in a female. At all events, we have the overwhelming preponderance of probability in our case.

Applying these remarks to our patient, he certainly shows from the earliest infancy a well-marked tendency to bleed profusely for long periods of time, from utterly trifling causes or from no ascertainable cause at all. As to the hereditary part, let me read the family history which, in some ways, resembles a fairy story. The patient's mother has had eleven pregnancies:

1. A full-term normal male who was circumcised on the eighth day of life. One hour after circumcision he began to bleed and the bleeding persisted in spite of strenous efforts to control it. He bled for three days and expired.

2. A male child who bled to death at the end of six weeks after a trifling injury.

3. A male child who was circumcised and bled for several days. The hemorrhage was finally controlled. At eighteen months the baby fell and cut his upper lip on his teeth, and in spite of skilful medical attention the baby bled to death.

4. Again a male child. Again circumcision and again the fatal outcome through hemorrhage.

5. The fifth child was our patient.

6. A female infant who was killed by accident, but did not die of hemorrhage.

7. A male infant, now twelve years of age. He is not a bleeder, was circumcised successfully and without undue loss of blood, and has since had his tonsils removed, also without undue loss of blood.

8. The eighth child was a male now nine years old, who was not circumcised. He got along very well until he was one year

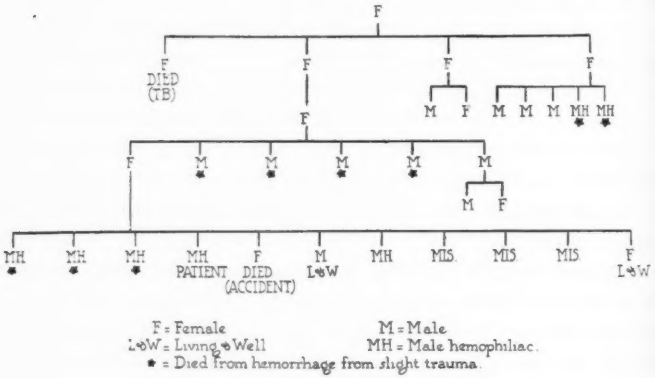

Fig. 17.—Family tree of hemophiliac.

of age, when he fell and broke a tooth. He bled for several days, and the hemorrhage was only controlled after his removal to a hospital and after great efforts had been made to check the bleeding. Many times since he has had serious hemorrhages from trivial wounds. He also has had hemorrhages into both ankles.

The ninth and tenth pregnancies were miscarriages, and the eleventh, now a two-year-old girl, is living and well with no hemorrhagic tendencies.

In addition to this the patient had four uncles and two cousins on the maternal side, all of whom bled to death after circumcision.

I think you will agree with me that with this familial history, and especially with the histories of his uncles being bleeders, that this patient must be ranked as one of the classical and striking hemophiliacs.

The nature of the disease is still obscure, and of all that has been known for many decades a very few points only are still established indisputably. The familial nature of the disease, the definite line of descent through the female, the occurrence almost entirely, if not quite so, in male children are points about which there is no dispute.

On the other hand, when we come to a study of the blood, the explanation for the striking hemorrhages is not quite so apparent. It is perfectly obvious that after repeated hemorrhages there will be a secondary anemia proportional to the extent of these hemorrhages.

Taking up a consideration of the blood itself the situation is as follows: The bleeding time is commonly stated to be normal. This, however, depends to a very marked extent upon the phase of the disease at which the determination is made. In our patient, taken by the usual method, it was considerably over one hour, and the patient was still bleeding ten and one-half hours after an ear puncture. This test was made during the bleeding phase. Twenty-three days later, at which time the patient was greatly improved, the bleeding time was four and a half minutes, that is, within normal limits. The coagulation time was still very long, namely forty-four minutes and the platelet count 230,000. In our patient the red blood-count was 3,800,000; hemoglobin 48 per cent. (Newcomer). During the bleeding phase the coagulation time was thirty-seven minutes using the Howell method. A normal control showed a coagulation time of twenty minutes. At the same time the prothrombin time was one hour, as compared with thirteen ninutes in a normal subject. The first platelet count was 250,000; the leukocyte count was 6600. Of these there was 78 per cent. polymorphonuclears, 10 per cent. small lymphocytes, 11 per cent. large lymphocytes, 1 per cent. large mononuclears. Numerous blood examinations were made from time to time, but the one just noted was fairly typical of all

the rest. It is especially worthy of note that the platelets did not go below 230,000 at any time. It is distinctly interesting to note that the tourniquet test was definitely positive in one and a half minutes, although it should be observed that this was only after he had been in the hospital some time. At the time this test was applied his bleeding time had been reduced to four and a half minutes.

A great deal of special blood study has been done on this patient, but as it is not essential to the diagnosis we will dismiss it with a brief mention. The volume per cent. was 0.79; the volume index 1.05; the saturation index 0.60; the color index 0.64. The technic used in these determinations was that of Haden. The fragility test showed that hemolysis began at 0.4 and was complete at 0.36. A normal subject, simultaneously tested gave exactly the same result. These tests were made during the bleeding phase.

Physical Examination.—We will confine ourselves to the points which are definitely positive and which have a bearing on our diagnosis.

The scalp, hair, eyes, ears, and nose are negative. The mouth is quite normal with the exception of very bad teeth, many of which are carious. This is due to the fact that he has been afraid to have them attended to because of the fear of hemorrhage. Tongue is clean; tonsils enlarged and cryptic. Neck is negative.

Chest: Heart and lungs quite normal in every respect, no murmurs, no thrills.

Abdomen: Negative. Liver and spleen negative.

Genitalia shows the evidences of circumcision. Inguinal glands just palpable. No other evidences of adenopathy anywhere to be found.

Extremities: Upper: Considerable discoloration due to extravasation of blood on the inner aspect of the right arm and on the left shoulder. Both elbows show marked limitation of motion, neither extension nor flexion can be accomplished completely. Range of motion about 100 degrees.

Lower: A glance at these shows that the left leg is markedly

shortened, being only 79 cm. long as compared with $84\frac{1}{2}$ cm. on the right side. Marked limitation of motion in the left hip and in both knees. In the latter the angle of flexion is about 45 degrees. There are multiple purpuric spots scattered irregularly over both lower extremities, varying in size from that of a pinpoint to that of a half-dollar.

Urinalysis: As noted in the history, blood is abundantly present off and on in the urine. The feces, however, have been entirely free from blood on several examinations.

Fig. 18.—Hemarthrosis in both hip-joints.

One or two other points might be noted. One is that the blood calcium is 9 mg. per 100 c.c. of blood. A second examination gave 1042 mg. for the calcium with 2208 mg. for the potassium.

The blood Wassermann is negative, as is also the blood chemistry.

The symptoms in this case are so classical that they need but little comment. In most cases the disease begins early in life,

occasionally not until the boy is half-grown or grown. Almost invariably the early symptoms are a replica of those in this patient, the first thing attracting attention being spontaneous hemorrhage or a hemorrhage from an insignificant cause. They may be from the stomach, intestine, kidneys, joints, nose, buccal cavity, or the skin, and often last as they did in this patient for days on end. Hematomas are common from very small injuries.

I would call especial attention to the hemarthrosis. The knees are the favorite place, and the hemorrhages occur either in the knees or one of the other large joints in a very large proportion of the cases. The subsequent history may be that the blood is either absorbed or, as in this patient, it produces an arthritis which may be erroneously regarded as of infectious or tubercular origin. Ankylosis is not very rare. As in other diseases, hemophilia is of varying grades. It is hardly necessary to say that in this family the grade has been most severe, since the majority of those affected by the disease have succumbed to hemorrhage, and this boy only escaped by a narrow margin the same fate.

A few words on the subject of diagnosis. The true hemophiliac begins his bleeding early. The hemorrhages are persistent and show a very special predilection for the joints, which may be almost destroyed. The coagulation time of the blood is greatly increased, and is said to be particularly so in the time between bleedings. We have already noted this in our patient.

The only disease with which hemophilia is likely to be confounded is hemorrhagic purpura, which is not hereditary, or at least if so, only by accident. It affects male and female patients alike, and has as its peculiar and characteristic symptom a great diminution in the thrombocytes or platelets. This brings with it a great increase in the bleeding time. In purpura hemarthrosis is not common, in hemophilia it is exceedingly common. In purpura the skin hemorrhages are very marked, in hemophilia much less so. That this last point is by no means a clean-cut diagnostic criterion is shown by this patient.

The outlook in a bleeder is, of course, pretty bad. In families which have been carefully studied something like half of them die in childhood, although dying in the first hemorrhage is reserved

for the especially severe cases, such as this family. It is, however, true that with each succeeding year the tendency becomes somewhat less, and so it is possible for the bleeder to grow up and enjoy a very fair degree of health.

You will remember that I referred in speaking of the mother's pregnancies to the fact that the eighth child, now nine years old, has also had many serious hemorrhages from trifling wounds. Furthermore he has a hemarthrosis in each ankle.

While this child has not been in the hospital as a patient, we asked the mother to bring him in so that we might check up on his blood condition. This, of course, was not in the bleeding phase. The results were as follows: Erythrocytes, 3,300,000; leukocytes, 10,700; platelets, 270,000; hemoglobin, 55 per cent. (Newcomer). The coagulation time (Howell's method) showed a surprisngly long period, fifty-nine minutes. A normal simultaneously taken showed a twenty minute coagulation. The prothrombin time was two and a half hours; bleeding time five and a half minutes. The percentage volume of cells, 0.95; volume index, 1.44; saturation index, 0.58; color index, 0.83. The fragility test was normal, hemolysis beginning at 0.4 and complete at 0.36.

Of the above the only items which are of diagnostic significance are the great prolongation of the coagulation and prothrombin time with a normal bleeding time and platelet count. Taken with the history of repeated bleeding of long duration, they are quite sufficient to establish the diagnosis of hemophilia in this child as well as in his brother.

Nature of the Disease.—Relatively little is positively known on the subject except that there is a congenital inability to produce a proper thrombin. It may, therefore, be classed as a ferment-deficiency disease, relative in character, in which the clotting mechanism is insufficient on the organic side. It is not due to a lack of calcium salts.

Treatment.—We have used intramuscular injections of fresh human blood in 25 or 50 c.c. amounts. These may be repeated twice every twenty-four hours, if necessary. For uncontrollable hemorrhage transfusion is probably the most satisfactory.

CLINIC OF DR. ISAAC A. ABT

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EXAMINATION OF THE INFANT. SPECIAL CONSIDERATION OF ABDOMINAL PALPATION

BY special training and long experience the pediatrician becomes especially adapted to recognize disease in infants and children; he acquires knowledge of their disorders; he becomes an adept as a general practitioner in the diagnosis and treatment of diseases of children.

Finkelstein, in a recent publication, speaking of the personal fitness of the children's doctor, says: "One doctor acquires at the outset the correct method and proceeds with unfailing success; the second learns the art after long practice; and the third never acquires the art."

I presume that is true of almost everything in life.

To succeed in the examination and in the treatment of children one must have a refined quality of human feeling, a keen sympathy, friendship, and love for the tiny being; and more than all, he must know the vagaries, the weaknesses, and psychology of mothers. He must understand the elemental pedagogic and psychologic procedures, so that he may counsel concerning the training of the child, interpret and advise the mother without causing resentment or unpleasant reaction.

Formerly the management of the diseases of infancy and childhood was a by-product of the obstetrician and internist. In more recent times when the study of children became a special object of interest, clinicians and laboratory workers were stimulated to make special researches, and the beneficial results are too numerous to be told in a brief record. No other department of medicine has made greater advances, nor has any other department contributed more to the total of human happiness.

I wish to refer to one or two points in the physical examination of infants and children. As a teacher of medicine, more particularly of pediatrics, for more than thirty years, I frequently have the feeling that as doctors we are wont to forget our historic consciousness, if such exists. Medical knowledge should not consist of the novelties which have appeared on the printed page the last month, or year, or decade. Valuable observations in the diagnosis and course of disease as well as in the treatment were known to our ancestors. We have, it is true, perfected to some extent the discoveries of Laennec and Skoda in physical methods of auscultation and percussion, but we should not forget the debt which we owe to them and what they have taught us.

The intern or recent graduate is apt to begin his examination by making a chemical blood test, or a Wassermann reaction, or a spinal puncture test, or by determining the metabolic rate, or by making an *x*-ray examination. It is obvious that this type of procedure is fallacious. The patient's history should be obtained and then the patient should be examined. The examiner should exercise to the utmost his special sense organs. He should inspect, and the image which he receives should convey a real meaning, or at least should receive an interpretation. He should percuss gently and auscultate attentively. He should receive such information as comes to him from his sense of smell. We are informed that the older doctors recognize smallpox and typhoid fever by characteristic odors with an unfailing accuracy. Gangrenous diphtheria and noma are revealed by the odor, and an acetone breath may be the first suggestion of diabetic coma.

Lastly, the examiner should touch and feel. Feer, the distinguished professor of pediatrics at Zurich, writes in his book on diagnosis, that he requires from his young assistants a record of physical findings before an instrument is used or a pinprick wound inflicted. I may add, after one has made his observations, and if circumstances permit, the physician should be allowed a few moments to sit quietly and think, because, after all, one should sufficiently compose himself so that he may put all his findings together and interpret them in the light of his knowledge and experience. Diagnosis is a thinking act, not a machine-made

process. Most precious of all is good common sense and ordinary judgment which very often help the physician to make a very reasonable diagnosis.

After the preliminary interviews with the mother have terminated, the baby should be presented for inspection. If asleep, the baby should be viewed in its little bed; if awake, the baby should be undressed and held up for inspection. The experienced physician obtains much valuable information by mere inspection. A keratitis, hydrocephalus, rickets, scurvy, idiocy, infantile cerebral palsy, and a great variety of diseases are revealed by simply looking at the patient.

The severity of the illness is frequently recognized. The frequency and character of respiration will often indicate a story and enable one to make a presumptive diagnosis, which may be confirmed by more extended physical and laboratory examinations.

The history of auscultation and percussion has been made familiar by the writings of numerous authors. The technic of these methods is amply told in every text-book dealing with the subject.

Palpation.—I wish to refer briefly to one of the most frequently employed and probably the oldest of all methods of physical examination, namely, palpation. In examining historic records one finds no account of the discovery of this method. Like inspection, it is a natural, almost an instinctive, art. One looks, then feels, if his curiosity is sufficiently aroused.

The ancient physicians describe the perceptions obtained through the sense of touch. A mass was hard or soft or fluctuating one learns from Hippocrates. A phlegmon was hot or pulsating. Therefore we conclude that the art of palpating is as old as the race of physicians who have practised in the civilized world.

In the eighties of the last century the knowledge of palpation included almost exclusively those conditions which prevailed in tumor diagnosis, and how primitive these diagnostic methods were may be realized from an expression quoted from Billroth, who said that whenever he opened the abdomen for an

acute condition he was happy if he met an abdominal affection instead of a pneumonia or thoracic disease—a condition of affairs which is confirmed by other writers of Billroth's period.

The same thought is expressed by a Russian surgeon, Slifossowski. When asked to which organ a tumor belonged, he replied, "It belongs to the abdomen."

Later on the internists awakened to the desirability of making preoperative diagnoses, and they attempted to apply palpatory methods in the recognition of abdominal tumors. Among those who made pioneer studies in palpation were Rosenheim and Minkowski, though it should be acknowledged that most internists made their diagnosis of intra-abdominal conditions after exploratory laparotomy had been performed.

Surgeons, as a rule, were in the same position as the internists with reference to abdominal diagnosis. The first one who attempted to systematize and organize knowledge in this direction was a German surgeon, B. Pagenstecher. He described the palpatory peculiarities of tumors of various abdominal organs. He also was the first to inflate the stomach and intestines in order to localize tumors. Up to this time no one had perfected palpatory methods in the diagnosis of abdominal conditions or in the disorders of the alimentary tract, other than the method used for detection of tumors.

Glénard and Obrastzow were the first who attempted examination of the colon by palpatory methods, and Hausman was the first to elaborate them for practical use. According to Hausman, the cecum, the appendix, the pylorus, the greater curvature might be felt under certain circumstances. If your reaction is the same as mine when I first encountered these writings, you will say under your breath, "I doubt it." But wait. He claims to have done this, to have palpated these various abdominal viscera.

About this period Glénard recognized for the first time that nephroptosis was a part of general visceroptosis. He also noted that the colon moved with respiration and could be passively pushed upward and downward, but not laterally. He also was able to show that the transverse colon could be differentiated

from other organs in the abdominal cavity, such as the stomach, pancreas, or intestines.

Glénard's observations were not taken seriously, or at least were not accepted by his contemporaries.

Obrastzow, however, worked independently of Glénard, and confirmed and amplified his findings. Obrastzow succeeded in palpating the normal colon in a considerable number of cases. He could feel the transverse colon in 33 per cent. of cases, the cecum in 53 per cent., and the sigmoid in 40 per cent. Obrastzow's conclusions were not generally accepted. Most of the textbooks on internal diagnosis do not mention his method.

Hausman, in discussing the findings of Glénard and Obrastzow regrets that so few have been willing to accept these methods in routine abdominal examination. He thinks that physicians would be more ready to receive a new laboratory method than one that could be so easily applied as palpation, and particularly abdominal palpation.

I have no doubt that the ability to palpate well is to a large extent an individual endowment. The sense of touch and the muscle sense will explain the genius of the virtuoso, who has shown in his early childhood, inherited ability at the piano or with the violin or other muscial instrument.

The same refined muscle sense probably explains the talent for drawing, painting, sculpturing, or, indeed, anything which requires touch or muscular movement. On the other hand, the sense of touch and the dextrous use of small muscles may be acquired, notably the blind learn to read by using raised letters. There are numerous instances where children of mediocre talent are taught to paint, draw, and mold in clay. It seems, therefore, that the ability to palpate depends to a degree upon the natural dexterity and the inherited delicacy of touch and muscle sense. On the other hand, the art of palpation may be acquired by nearly every one by diligent and thoughtful practice.

Obrastzow and Hausman have formulated some general directions. It is important that the examiner should be sitting in a comfortable position. The familiar advice appears in every text that the examiner's hands should be warm. The patient should

be in the recumbent position. Hausman advises that the head be pressed deeply into the pillows. The legs should be outstretched. It is of no advantage, the best authorities say, to have the thighs flexed on the abdomen. The difficulty in abdominal palpation is increased by the interference of the drawn-up thighs even if in some instances there is complete relaxation of the abdominal wall.

Hausman has suggested the method of *gliding* and *deep palpation*. By adopting this procedure he claims he is able to palpate the large intestine, and also to recognize portions of the stomach. In certain instances he can even feel the appendix.

The most important factor is overcoming the resistance that is offered by the abdominal wall. Great force should not be used. Indeed, forceful efforts do harm and affect the procedure adversely, because they cause muscular contractions and increased resistance.

The utmost attention should be given to the respiratory phase. During inspiration, particularly in abdominal breathing, the muscles contract and the resistance increases. The important point is that in performing deep palpation the fingers should come to complete rest during inspiration. At the moment when inspiration ceases and expiration begins the maximal relaxation occurs. During this period of relaxation the fingers may be pushed deeply into the abdominal cavity, though the manipulation should not be too energetic or too rapid. It should be stated, however, that the maximal resistance may vary in some individuals. In rare instances it may occur during the inspiratory phase. In every case one should find the relaxing period, and this phase should be used at the opportune time or moment.

Hausman gives the following directions for palpating the greater curvature of the stomach which may serve as an example of his technic: He introduces one or more fingers into the epigastric region, attempting to penetrate as deeply as the presenting surface of the spinal column. Then he makes sliding movements from above downward, that is, in a transverse direction to the greater curvature. These sliding movements are made in rapid succession in both directions, that is, from above downward

and from below upward. They must be continued until the fingers are felt to sweep over the greater curvature, The deep palpation should be conducted during the expiratory phase. With a similar technic Hausman claims to have palpated the pylorus in adults, also the swollen or hypertrophied pylorus in children.

Several cardinal points are stated which express Hausman's technic of palpation. The manipulation should be conducted first, in the right direction; the long axis should intersect at right angles the area of the digestive tube to be palpated; second, in the right place, that is, where the suspected region is located; third, at the proper depth; fourth, at the right time, that is, during expiration.

Eliciting tenderness is an important factor in palpation. There has been much discussion as to whether the abdominal pain and tenderness are produced by inflammation in the deep-lying organs or whether they are purely cutaneous according to Head.

Deep palpation is usually impossible during an acute attack of appendicitis or any acute infection of the abdominal organs. In some cases of acute appendicitis deep palpation may be performed if a relatively slight amount of force is used, and, if one proceeds cautiously, without producing great pain.

In the so-called chronic appendicitis palpation has more value as a diagnostic measure. Hausman maintains that it is sometimes possible to feel the diseased or enlarged appendix, and also to elicit deep-seated tenderness which may be most marked in the ileocecal region, though occasionally the pain is felt in other places, such as the hepatic region, the epigastrium, the left side of the abdomen, or even in the back.

It need hardly be mentioned that palpation at first should include the superficial portions of the abdomen, and finally the deeper portions should be explored.

Distention of the abdominal cavity with gas or fluid, the accumulation of food in the stomach, the pressure of fecal masses in the large intestine, or the distention of the bladder with urine are sufficient at times to defeat the most expert in palpation.

In nervous adults or in children in whom the slightest touch reflexly causes contraction of the abdominal muscles, Obrastzow recommends that the examiner should press on the side opposite the one to be examined. This procedure causes the contraction of two areas; the site which is to be examined tends to become relaxed somewhat and enables the examiner to press more deeply into the abdomen. As a matter of fact, this manipulation does not always succeed.

It is sometimes difficult to differentiate voluntary contraction of the abdomen from contraction of the muscle due to intra-abdominal inflammation. In the latter case the muscles stand guard against the production of pain. Finkelstein recommends that the volar surface of the middle finger be placed on the abdomen in various regions, and with the middle finger of the other hand pressure be made over the nail of the palpating digit. Pressure should occur at the moment of expiration, and light vibratory thrusts should be made. In this way the various regions of the abdomen are investigated. In those instances where the muscles are standing guard no perceptible impression is made on the rigidity. Where the contraction is due to fear or nervousness, the muscles yield on moderate pressure.

It may be necessary, especially in children, to anesthetize in order to effectually examine the abdomen. Palpation by rectum in children as well as in adults may yield valuable information, particularly if the lesion is located in the pelvic region.

Gubergritz suggests that the patient's position be changed, especially in difficult cases. For example, if there be considerable ascites and it is desirable to examine the liver, the patient should be turned on the left side. In this position one frequently succeeds. Israel suggests that if the left kidney is to be palpated the patient should be placed on the right side. He says that the kidneys approach the midline and descend slightly and may be palpated in this position. Similarly, the spleen may be felt if the patient is lying on the right side.

Gubergritz found that if he wished to palpate the right hypogastric region he met with considerable success if he placed the patient on his left side. He thinks that not only the ileum,

cecum, ascending colon, and appendix may be felt in this left lateral position, but also the sigmoid and descending colon, under some circumstances. He also claims that patients suffering from chronic appendicitis give no evidence of tenderness when examined in the dorsal recumbent position, but if the same patient is examined in the left lateral position pain may be elicited to the right of the right rectus muscle. Rosenstein in 1922 also believed that appendiceal tenderness was more easily elicited in patients who were placed on the left side. Gubergritz also reports the examination of patients in the intervals between attacks of biliary colic. Those who were examined in the dorsal recumbent posture showed no tenderness, while in those who were examined in the left lateral position tenderness was elicited in the liver in the majority of cases.

Palpation of the abdominal organs is an art that should be learned and taught. One who has a skilful hand and who reflects and thinks about his findings will make the most accurate diagnoses.

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CLINIC OF DRS. FRED M. DRENNAN AND FRANK C. VAL DEZ

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SIMPLE ULCERATIVE COLITIS

THE condition variously known as simple ulcerative colitis, idiopathic ulcerative colitis, innominate ulcerative colitis, etc., has been recognized for a great number of years, but has seemingly not had the attention from internists that its severity and high mortality should justify. Wilks in 1875 and White in 1888 were the first to give definite consistent clinical pictures and pathologic findings. Logan, after a lapse of years to 1919, and Lynch, 1925, also give a clear concise statement of symptomology and suggest various methods of treatment, all of which, they agree, are not satisfactory. The high mortality, varying from 7 to 35 per cent., and the much higher morbidity resulting from ulcerative colitis makes anything that can add to our knowledge or method of treatment very much worth while.

Before taking up specific suggestions it might be well to review the clinical and pathologic aspects of the condition. It is described as a chronic inflammation of the large bowel of unknown etiology, acute or of long standing, and showing all grades of inflammation from reddened, congested mucosa to superficial and deep ulceration. In reviewing the literature and case histories of our own cases the following facts seem to be definite:

1. That onset is usually in the young adults—twenty to forty years—a very few cases in children and in older people. Sex, occupation, and family seem to play no rôle; locality must be considered in view of the possibility of amebic etiology for the ulcerative condition. Occupation only as it may influence exposure, as various authors speak of circumstances in which a

number of people eating the same type of food may have severe diarrhea, but only one person have an ulcerative colitis resulting.

The pathology varies due to the severity of the infection, the resistance of the patient, and the stage of the disease at which the examination is made. Early in the course in normally nourished individuals the mucous membrane is red, congested, granular in appearance, bleeds easily on slight trauma. This progresses, the mucous membrane breaks down in small patches, becomes necrotic and sloughs off, leaving an ulcerated area, usually with the submucosa as a base. The fresh ulcers usually are clean cut, edges not undermined, they vary in size, and have a tendency to remain superficial. As the disease progresses they tend to coalesce, giving ragged irregular mapped out areas with portions of intact mucosa between them. If this intact mucosa becomes edematous, it assumes a polypoid appearance. The microscopic examination shows first an acute inflammatory process, the mucosa being congested, having small hemorrhages, being infiltrated with leukocytes and plasma-cells; later there is necrosis, small round-cell infiltration, extreme hyperplasia, and fibrosis, which gives the typical tube-like condition of the gut. In the simple ulcerative colitis the lesions usually are most severe and of longest duration in rectum, gradually extending to the cecum, while in the amebic type the primary lesions are supposed to be in cecum extending downward. Logan speaks of two types: 1, A chronic slowly developing type; 2, a rapidly developing type associated with marked severe symptomatology, the difference perhaps being due to the resistance of the patient.

The clinical picture varies also with the type of individual attacked. The usual acute case shows the following symptoms:

1. Diarrhea—usually severe—three to twenty or more stools daily; most of them in morning; usually persistent, though at times patients have alternating constipation and diarrhea. At times onset may be more gradual with patient first noticing:

Blood and *mucus in stools*, the blood varying from dark brown to red color; if bowel movement formed, red blood may be on outside of such particles.

Stools are of very foul, putrid, offensive odor, full of pus,

blood, and mucus; occasionally shreds of sloughing tissue; they are alkaline in reaction, and unless diarrhea is excessive food has been fairly well digested.

Pain, cramp-like, colicky type; worst p. c. and associated with bowel movement; relief by bowel movement or passing flatus. Frequently there will be severe urgency and rectal tenesmus, giving almost continuous desire to stool. Incontinence is not unusual in severe cases, especially in debilitated individuals.

Abdominal Tenderness.—Present along the course of the colon; not marked save where near perforation with localized peritonitis or abscess formation is present.

Nausea and *lack of appetite* may be frequent, but vomiting is not the rule.

Loss of Weight.—Constant; in few conditions it is more marked, the dehydrating diarrhea, lack of appetite, pain, etc., all contributing.

Anemia at times severe; of secondary type, though at times it approaches a primary pernicious type, the patient having a peculiar waxy pallor.

Temperature.—Usually slight temperature and leukocytosis present.

Certain severe cases show iritis, neuritis, joint involvements, erythema nodosum, etc.

x-Ray examination shows a tube-like colon with haustration gone in the areas involved; severe and prolonged spasms and areas of permanent narrowing and thickening. There is also rapid emptying, the barium acting as a definite irritant.

Proctoscopic examination should be made with a certain amount of caution because of the possibility of injury and perforation. It shows a markedly congested granular mucosa which bleeds readily on slightest trauma. Numerous small pin-point ulcerated areas, some large areas, and in severe cases profuse necrotic sloughing areas.

Treatment in ulcerative colitis, as we stated in a previous discussion, has varied greatly, there being practically as many methods as there are authors on the subject. From the diet standpoint some men have held to a strict milk diet; others

have eliminated milk entirely; likewise, all cereals or all meat diets have had their ardent followers and just as ardent opponents. It seems it should be granted that a nourishing non-irritating diet, as near cellulose free as possible, with green vegetables and fruit eliminated, is the most rational type of food, getting in sufficient quantity to cause a definite gain in weight, strength, and general resistance. Local application of heat gives relief to some gut spasms and lessens frequency of bowel movements.

Rest is granted by all as being definitely important.

Medication.—No disease (as one author states) has been blessed with so many healing drugs, and, so far as we know, no disease has so successfully resisted all such medication. The list includes many bismuth preparations, bismuth, and chalk of Sippy management, various silver preparations, vegetable extracts, as the Tannigan group, Kolin, opium preparations, probably the best in quieting the excessive peristalsis, iodin of Logan, etc. Likewise the local treatments by irrigations have varied, including simple boric acid, sodium, and potassium permanganate, salicylic acids, zinc sulphate, silver salts, quinin, iodin, neutral acriflavin by Crohn, etc. All irrigations have the difficulty that if weak solutions are used no reaction is secured, while if strong enough solutions to be bactericidal are used they are definitely harmful to the mucous membrane and cause markedly increased pain to the patient. Einhorn uses a small tube which is passed to the cecum and 10 per cent. gelatin enema with 10 to 15 drops of 1 : 1000 adrenalin is used. Sippy used a bismuth subgallate in mucilage of acacia as a retention enema with very good results.

Surgical treatment has varied, iliostomy, appendicostomy, partial or complete removal of colon have been done, the most rational being the measures which attempt to put colon at rest. After surgical treatment irrigations have been more regularly used because of the ease of manipulation.

It will be noted that the incidence of infection, the symptomatology, practically all the pathology as given, except the organism, could and would be found in ulcerative condition due

to ameba as it would be if the condition were due to any one of the various organisms regularly found in the colon, most all of which at various times and by various men have been held to be responsible for ulcerative colitis, or if due to organisms secured by scraping the mucosa, as suggested by Hawkins in 1909, or by securing material direct from the ulcer, as carried out by Bargen in his work, isolating a Gram+ diplococcus from which he has made an autogenous vaccine which is to be given in proved cases. This idea plus the fact that several authors have suggested the possibility that the so-called simple ulcerative colitis was caused primarily by the ameba, which has periods of comparative quiescence during which time secondary invading organisms would be found in the lesions, plus the fact known to any one who has looked for ameba that he may search for days without finding a single organism, and then find a specimen loaded with them, or have the embarrassment of having some one else find the organism on the first examination, caused us in our work at Mercy Hospital to examine particularly carefully all cases for possible amebic infection even though it was originally felt that the probable diagnosis was simple ulcerative colitis.

We began our observations early in the summer of 1925, the data being first given at the Calumet Branch of the Chicago Medical Society in September, 1925. At about this same time Myer and Hirsch, working in the Pathological Laboratory at St. Luke's Hospital, reported a series of 5 cases diagnosed clinically as idiopathic ulcerative colitis which at autopsy were proved to be amebic infections, giving added grounds for the idea of amebic dysentery being more frequent even in patients who have always lived in the Chicago area than we have been led to believe. During the two years we have had a series of 35 cases which we have been able to follow accurately. In all of the cases we have worked out the clinical history as outlined above, have made careful *x*-ray examinations, proctoscopic examinations with attempts to isolate organisms from material removed from the ulcerated areas. We have ruled out tuberculosis, malignancy, etc., in all cases, but have not used the cultural methods of Bargen because we felt that his work is such a special and accurate type

that we would not want to attempt it without having his technic under accurate control.

We have divided our cases into two groups: First, those in which we were able to isolate the ameba from material taken from the ulcers at proctoscopic examination; second, those which showed the same clinical picture, *x*-ray and proctoscopic findings, but in which the ameba could not be found. Practically all these cases were chronic in type and had been examined in other clinics where careful work also had failed to show the ameba.

At about the time of the beginning of this study we heard Dr. F. M. Johns give his paper at the American Medical Association (1925) on the Use of Stovarsal in Amebic Dysentery. Stovarsal, as you know, is an acetyl-amino-hydroxy-phenylarsenic acid containing 27.11 to 27.4 per cent. arsenic. It has a low toxicity and seemingly can be given without any fear of bad effects if one watches for arsenic rash and poisoning. It is safer than continued emetin medication, and gave us a drug which we felt could be used therapeutically in these cases which gave the clinical picture of an amebic dysentery, but in which the organism had not been found during a definite period of careful observation.

In both the first and second groups of cases the same routine of treatment was carried out—stovarsal, gm. 0.25, given three times daily for six to eight days. A period also of six to eight days' rest, and then another series given, etc. etc. About every six weeks a series of emetin injections, 1 gr. daily, have been given to cases where symptoms have cleared up slowly or where patient has had a return of pus and blood in bowel movement. Diet has been of the bland, nourishing type, but not markedly restricted. Tonic medication in the form of iron and arsenic preparations have been used, and when necessary opium in sufficient doses to help control diarrhea, has been used, but in all cases we have attempted to confine the treatment to anti-amebic medication. In 2 cases the symptomatology has continued so that even though there has been some improvement in general condition we do not feel results are good. In all the other cases we have been agreeably pleased with the results and have been

struck by the marked similarity in reactions. Let me cite 2 cases: One an intern in Mercy Hospital. Temperature 100+° F. each day. Four to eight loose watery bowel movements daily filled with pus and blood. Marked weakness. Severe abdominal cramps even with 60 to 80 drops of deodorized tincture of opium daily. On fourth day of stovarsal bowel movements became formed, pain much improved, general condition improved; at end of ten days stools free of pus and blood, and now after two years he continues O. K. At no time was ameba found. Second, a man from Texas who traveled all over the South, gave a history of diarrhea for years, pus and blood in bowel movements, marked tenesmus, cramps, with twenty to thirty bowel movements daily at times. Ameba found at first office examination. Within first seven days of stovarsal bowel movements became formed, two to three daily, pain less, general condition better, and with second series stools became free of pus and blood. These are, of course, the most striking cases of the series, but in all the good results have been definite. Some have required longer period of treatment, the shortest three months, the longest about fifteen months (in this case because of recurrences). We have had no deaths or serious complications.

In conclusion, we want it understood that in suggesting a therapeutic test in cases where laboratory findings should be exact, we do not want you to be any less painstaking in your effort to determine the etiology in each case, but when we consider the difficulty in finding ameba in certain cases, the possibility of lesions being secondarily infected, the similarity of pathology and clinical picture, and when we have a method of treatment possible which does not jeopardize the patient, I feel we are justified in trying this therapeutic measure, and if it influences any of the so-called idiopathic ulcerative colitis cases it is worth while.

CLINIC OF DRS. JULIUS H. HESS AND JOSEPH K. CALVIN

MICHAEL REESE HOSPITAL

ALBUMINURIA IN CHILDREN

WE wish to present to you today 6 cases whose common finding is albuminuria in order to illustrate the various clinical types which may be associated with this urinary finding in children.

Case I.—Growth or Developmental Albuminuria.—This patient is a girl aged nine years, in perfect physical condition, with normal weight, height, and development. Her physical examination is essentially negative. There are no obvious foci of infection. The urine, both A. M. and P. M. specimens, have shown + to ++ albumin intermittently for the past three years. For intervals of several months the urine may remain negative. The microscopic findings have always been negative. The blood-urea taken at intervals has varied from 10 to 15 mg. per 100 c.c. of blood. Her blood-pressure is 90 systolic and 60 diastolic. The hemoglobin is 85 per cent. and leukocytes 7000. The albumin content of the urine is not influenced by diet (either high or low protein) or any other form of treatment.

This type of case, in the absence of any positive findings except the albuminuria, we group as a developmental or growth albuminuria. About 10 per cent. of healthy boys and 20 per cent. of healthy girls show this condition intermittently, the incidence becoming greater as the period of greatest growth (puberty) approaches.

The condition is usually discovered accidentally in the routine examination of urine. Treatment is not necessary. The condition should be explained to the parents as a benign one and

the child instructed to lead a normal life and thus avoid developing albuminuria neurotics in the parents and child. Of course they should be kept under observation until the physician is convinced that he is not dealing with a pathologic kidney.

Case II. Malnutrition with Albuminuria.—This patient is a boy aged six years. At present this urine is negative and has been for the past five months. He came under observation about seven months ago. At that time his urine showed + to ++ albumin on repeated examinations, but negative microscopic findings. He was markedly underweight, malnourished, anemic, asthenic, had hypertrophied and infected tonsils, adenoids, and several abscessed teeth. Every physician sees numerous children of this sort, especially in dispensary and hospital practice. His blood-urea on several examinations varied from 12 to 14 mg. per 100 c.c.; the hemoglobin was 60 per cent.; the leukocyte count was 14,000; 60 per cent. polymorphs indicating an absorption from infected foci. His blood-pressure was 85 systolic and 55 diastolic.

The treatment instituted in this case was the removal of the infected foci (abscessed teeth, tonsils, and adenoids). The malnutrition was corrected by a forced diet, tonics, and fresh air. The albumin disappeared rapidly and has not recurred. Preliminary to the tonsillectomy a low protein diet was instituted to take the load off the kidneys, but this did not influence appreciably the albuminuria. If the blood chemistry and blood-pressure are normal, no fear of a general anesthetic need be entertained.

Children of the malnourished type taken as a group have an incidence of 40 to 50 per cent. albuminurias which we feel are not true nephritis, but a hypofunctioning of the kidney as well as the other organs from a general malnutrition and anemia due to chronic foci of infection.

Case III. Orthostatic Albuminuria.—This patient is a boy aged thirteen years. As you can readily see, he is tall, slender, of the asthenic type, rather pale, but intelligent appearing, rather

the student than the athlete. His posture is not good; he has rounded shoulders, prominent scapulæ and a tendency to lordosis and scoliosis. He tells us that he tires readily, sweats easily, often has cold hands and feet. No apparent foci of infection can be found. His urine specimen shows +++ to ++++ albumin, but is microscopically negative. However, if we have him bring us a specimen of urine obtained before arising from bed in the morning and another obtained about midafternoon (every albuminuria case should present two types of specimens for examination) we find that the morning specimen is negative, while the afternoon specimen often shows ++++ albumin (heat and acetic acid test). The microscopic findings are usually negative, although occasionally casts are found—most often hyaline. Adding acetic acid to the cold urine causes a + albumin cloud to appear. This is especially characteristic of this type of case. This specimen which we are showing you now contains ++ albumin (heat acetic test) as you can see. It was obtained from this patient about a half hour ago. Since then he has been standing for fifteen minutes in the lordotic position with his head and heels against the wall and his back arched forward. This second specimen obtained after this position test, shows almost twice as much albumin. If he remained in bed in the prone position for twenty-four hours that twenty-four-hour-specimen of urine would show no albumin. His blood chemistry is normal. His blood-pressure is systolic 95 and diastolic 65, perhaps a little low. His hemoglobin is 75 per cent., and leukocytes 8000. Diet does not influence his urinary findings. His daily urinary output is normal.

This case represents a fairly typical "orthostatic albuminuria," the quantity of albumin varying with posture. Posture is the distinguishing characteristic between this type and that previously designated as growth albuminuria. It is benign and rarely develops into a nephritis, but produces many albuminuria neurotics because this type of individual is unstable to begin with.

Orthostatic albuminuria may begin as early as six years of age, but the incidence increases as puberty is approached, but

practically always ceases spontaneously by the eighteenth to nineteenth year. The sexes are about equally affected. The majority of cases occur in tall children with considerable lordosis, and probably pressure on the renal veins by the lumbar vertebræ in the upright position accounts for the albuminuria. The type of individual is usually a healthy, but not vigorous type, often neurotic and suffer from other vasomotor disorders, as profuse sweating, palpitation, easy flushing, etc.

The treatment in this case is not to restrict the diet, but to encourage the eating of a wholesome menu, and to advocate plenty of moderate outdoor exercises. In other words, the treatment is to overcome the asthenic neurotic type of individual, and alleviate his excessive worry concerning the urinary findings by assuring him of the benignancy of the process. Orthopedic correction of posture, especially the lordosis, frequently improves the condition considerably.

The above 3 cases are non-nephritic albuminurias and may be classified as benign types. They must all be observed over a period of years, but the danger of nephritis developing in them is probably no greater than in other individuals. They may be classified respectively as: 1, Developmental albuminuria; 2, malnutrition albuminuria; 3, orthostatic albuminuria.

From the foregoing cases it is obvious that albuminuria in children may be benign. We believe that it has far less significance than albuminuria in adults which usually indicate some degree of nephritis. If the diagnosis of a benign albuminuria is fairly certain, then the patient and especially the parents should be instructed that such a condition as albumin in the urine can be a relatively harmless affair in juveniles. A benign albuminuria should be regarded by the physician much as a functional heart murmur in that the case should be kept under observation for a period of years to be certain that progressive organic changes are not occurring in the kidneys. Overemphasis to the patient may lead to neurosis.

The next 3 cases which we will show are also albuminurias, but are of the nephritic type. They represent the three most

common types of nephritis met with in children. Nephritis in children is much more commonly acute than chronic, and when chronic progresses more rapidly to a fatal termination than in the adult. Primary cardiovascular lesions leading to chronic nephritis are almost unknown in childhood.

Case IV. Nephrosis.—This case was completely worked up in the Pediatric Service of the Research and Educational Hospitals of the University of Illinois.

This patient is a boy four years of age with the following history. He was perfectly well until six months ago when following an upper respiratory infection (within a week) the legs and abdomen began to swell. During the next two weeks, the swelling (edema) became very marked, the face, eyes, abdomen (ascites), legs, and arms becoming involved. The urine was scanty and highly colored during all this period. His appetite, bowels, and sleep were practically normal at that time. For the past six months the edema has been intermittent, at times almost disappearing, with an increased output of urine, and then recurring as severe as before with scanty urine. A low-grade fever has been present intermittently since the onset. No other symptoms of significance were noted. The past history was negative, as was the family history.

Physical examination at present reveals a white male child about four years of age with a pale pasty complexion, very edematous with the eyelids swollen shut; the abdomen is markedly protuberant and distended, and the legs and feet are edematous. The ears are negative. A slight mucous discharge is seen in the nose. (Roentgen rays of the nasal sinuses were negative.) The teeth are in good condition. The tonsils are hypertrophied and infected. The anterior cervical and submaxillary glands are palpable. The heart and lungs are negative. Considerable ascites is present in the abdomen, but no tenderness, rigidity, or palpable tumors. The scrotum is markedly edematous. The hands and arms and especially the legs, ankles, and feet are extremely swollen and edematous.

The urine is highly colored (yellow), the specific gravity

averaged 1.015; the albumin is ++++; there are a few coarsely granular casts, few pus-cells, no blood. Repeated examinations have revealed *no blood;* 3.5 mg. of NaCl were present per cubic centimeter of urine.

The blood-counts indicate a marked secondary anemia, the hemoglobin ranging around 50 per cent. and the red cells 3,500,000. The leukocytes are 13,700, with 42 per cent. polymorphonuclears and 55 per cent. small lymphocytes.

The blood chemistry findings are important. The urea determinations averaged 18 to 20 mg. per 100 c.c. of blood, the non-protein nitrogen 35 to 40 mg., the uric acid 4 to 5 mg., the creatinin 1.5 mg. The cholesterol averaged 550 mg. per 100 c.c. (normal is 140–170). The chlorids as NaCl averaged 560 mg. per 100 c.c. of blood (normal is about 500). The blood calcium was 10.44 mg., which is normal. The total blood proteins (serum albumin and serum globulin) comprised 4.4 to 4.6 per cent. of the blood instead of the normal 7 or 8 per cent. The serum albumin was .83 to 1.62 per cent. (normal 4.6–6.7 per cent.), the serum globulin 2.88 to 3.62 per cent. (normal 1.2–2.3 per cent.). Thus besides the blood proteins being reduced in total per cent., the proportions were reversed, the serum albumin, usually 60 to 70 per cent. of the blood protein, was here only 20 to 40 per cent., and the serum globulin, usually 30 to 35 per cent., was here 60 to 80 per cent. The water content of the blood was from 82.8 to 83.9 per cent. (normal 77–81 per cent.). The blood Wassermann reaction was negative.

The blood-pressure averaged systolic 105, diastolig 65; no tendency to increase during the course of the case was noted.

The basal metabolic rate was +18. The phenolsulphonephthalein tests (injected intramuscularly) showed 33 to 40 per cent. excretion within two hours. Because of the edema and poor absorption of the fluid from the tissues by the blood this cannot be considered a true measure of kidney excretory ability. The kidneys may have excreted 100 per cent. of the dye in the blood, as possible only 40 per cent. was able to escape from the tissues into the blood, so great is the affinity of the tissues for fluids in this condition.

The von Pirquet test was negative, but the Mantoux was positive.

This case represents fairly typically parenchymatous or tubular nephritis, termed by many "nephrosis." In our experience it is not nearly as common as acute hemorrhagic nephritis described in Case V. Pathologically the tubular apparatus of the kidney is mostly involved, various degenerative changes being present in the parenchymatous cells. No evidence of inflammatory reaction is present in the kidney, consequently the term "nephrosis." Many pathologists, however, believe that as epithelial cells can only react to inflammation by degenerating, and thus an inflammatory reaction cannot be ruled out, the term "parenchymatous nephritis" is justifiable. Marriott of St. Louis and others believe that nephritides are general diseases affecting the entire body as well as the kidneys, and that in the type under discussion there is a change in the permeability of the body cells and an alteration of the physical chemistry of the body due to a substance which changes the surface tension of the blood and the permeability of the renal tubules. Epstein and others maintain that the essential factor is a disturbance in protein metabolism, a sort of protein diabetes. Still others hold that the primary change is a lipemia and cholesteremia which eventually damage the kidney tubules with resulting albuminuria, etc.

Many of the cases apparently are due to a staphylococcus infection usually of the nasal accessory sinuses, which initiates one or all of the above metabolic changes. In our case no such infectious basis could be found, although the case began with an upper respiratory infection. As a rule the streptococcus types of infection, such as tonsillitis, cervical adenitis, and scarlet fever, are more prone to cause hemorrhagic types of nephritis (glomerular), while "colds," sinusitis, bronchitis, etc., predispose more toward producing nephrosis.

The symptoms of nephrosis begin insidiously, usually following shortly after an acute exacerbation of some chronic upper respiratory infection. Essentially, marked generalized edema, extreme pallor, and scanty urine containing large amounts of albumin, but no blood characterize the condition clinically.

The patient is usually comfortable and has a fair appetite, whereas in hemorrhagic nephritis with this degree of edema (a severe form) much prostration would be present. The blood-pressure is normal; there is no abnormal retention of non-protein nitrogenous products in the blood; the blood proteins are considerably reduced, and the ratio between the serum albumin and globulin reversed. The chlorids are much reduced and the cholesterol and fat much increased in the blood. The renal function tests are usually normal. The basal metabolism is often below normal. In our case the reading obtained was above normal.

In the more acute cases the edema may subside gradually in the course of two or three weeks after elimination of the offending focus of absorption. This is most often spontaneous. A moderate amount of albumin may persist for weeks later. In others neither the edema or albumin completely disappear, merely subsiding, and then recurring from time to time. Even in those apparently completely recovered exacerbations are very prone to occur with upper respiratory infections.

Uremia and acidosis do not occur and unless the patient succumbs to intercurrent infections (due to low resistance) he probably will eventually recover even after several years of exacerbations.

The treatment consists mainly in *eliminating foci of infection* (draining nasal sinusitis, etc.) if such can be found. It is our experience that in many cases no such focus can be found. When sinusitis is the cause, draining of the infected area results in a rapid recovery from the nephrosis. A high protein *diet* 2 to 4 grams per kilo, with 60 to 80 total calories per kilo in the diet tends to increase the blood protein, and decrease the edema and albuminuria perhaps by stimulating protein metabolism. *Thyroid extract* aids in the same manner. *Salt* should be restricted. *Fluids* need not be restricted unless the edema is so great as to interfere with respiration and cardiac action. Water often acts as a diuretic. Sweating and purgation are of doubtful value. This case was receiving $MgSo_4$ over a period of several weeks.

For temporary symptomatic relief of the edema the use of

purine diuretics are of distinct value and do no harm, although the fluid reaccumulates when they are discontinued unless the underlying cause is removed. In the present case 10 gm. of urea given daily in cereal produced a marked temporary diminution in the edema. Other purine diuretics are caffein and theobromin sodium salicylate (diuretin). If the ascites is excessive, abdominal paracentesis is indicated.

Case V. Acute Hemorrhagic Nephritis.—This little girl, aged seven years, entered the hospital two weeks ago. The following accurate history was obtained from her intelligent mother: The patient had two rather severe attacks of tonsillitis each winter for the past three years. The last attack occurred three weeks ago. Five days after the onset of the attack, after the temperature had been normal for forty-eight hours and the little girl was feeling well again, there was a sudden onset of another group of symptoms. A slight rise of temperature occurred, the child exhibited anorexia, malaise, and lassitude, but did not appear severely ill. The most striking symptom was the appearance of blood in the urine causing it to look reddish and smoky. The quantity was considerably diminished. Two days later the child was admitted to our hospital clinic and briefly presented the following findings: A fairly well-nourished little girl with a rather pale puffy face, the only evidence of edema being a moderate puffiness of the eyelids and very slight pitting about the ankles; the patient was bright and cheerful and did not seem acutely ill; the temperature was 100° F. The tonsils were large, cryptic and red, and the upper cervical glands were readily palpable. The heart action was exaggerated (forceful apex-beat and loud sounds), but not enlarged, and no murmurs. The pulse rate was 90 per minute. There was no tenderness over the kidney or bladder region, or were the kidneys palpable. Nothing else of significance was found on physical examination.

The amount of urine passed in twenty-four hours had almost returned to normal (the initial suppression was very temporary), but it still appeared red and smoky. Albumin ++ (moderate amount) was present. Microscopically, erythrocytes were

abundant, leukocytes fairly numerous, and granular casts present in very moderate numbers. The blood showed a marked secondary anemia, hemoglobin 60 per cent., erythrocytes 3,800,000; leukocytes 14,000; 70 per cent. polymorphonuclears. The urea nitrogen in the blood was 25 mg. per 100 c.c., the creatinin 1.6 mg. The blood-pressure was 120 mg. systolic and 75 mg. diastolic (a distinct increase over normal for seven-year-old child).

The family history and past history of the patient showed nothing of significance except the history of repeated tonsillitis mentioned before.

During the past two weeks the blood and albumin in the urine have diminished considerably, the blood only being observed microscopically (the urine appearing clear grossly). The albumin is now only + and only occasional casts are seen. The amount of urine excreted is normal. The blood-pressure is normal, systolic 90, diastolic 60 mg. the blood-urea is again normal at 14 mg. The secondary anemia persists and the face still appears pale and puffy, although no obvious edema is present. The intradermal injection of a drop of normal saline remains partially unabsorbed for over one hour, indicating the absence of a tendency toward edema.

Unless unforeseen complications arise, this case will go on to a complete recovery within the next month or so. It represents a prototype of the most common form of nephritis seen in childhood, namely, acute hemorrhagic (glomerular) nephritis in which there is a general capillary damage throughout the body due most probably to streptococcus toxins; the kidneys having very rich capillary beds in the glomeruli, which must excrete most of the streptococcus toxins, are most affected by this capillary damage. Of course the tubules and interstitial tissues are secondarily affected to a more or less extent.

In the average case the onset is sudden following an acute streptococcus infection (tonsillitis, scarlet fever, etc.), but without much prostration, the attention first being directed toward the kidneys by the bloody urine. Edema may be moderate or even absent. The non-protein nitrogen in the blood is increased

as is the blood-pressure early in the disease. Occasionally, however, a more severe form is encountered, ushered in by impending uremia or uremic symptoms and accompanied by considerable edema. In these cases probably a more diffuse involvement of the kidneys occur.

Nearly all of the average cases (90 per cent.) recover in from one to three months, the albumin being the last to disappear from the urine. Recurrence of blood may occur with any acute infection within several months of the onset of the original condition. About 10 per cent. become chronic. Death during the acute stage usually results from uremia or acidosis.

The treatment consists first in prophylaxis, that is the removal of foci of infection before they cause a nephritis and the protection of the kidneys during acute illnesses especially of the streptococcus group. Such protection consists of partially resting the kidneys by giving a low protein diet, prevention of acidosis by giving plenty of carbohydrates, diluting the toxins by plenty of alkaline fluids, stimulating elimination through the skin and bowels by sponging or bathing and mild laxatives respectively, protection against chilling of the body, sufficiently long rest in bed, and repeated urine examinations.

The active treatment consists in the following: A low protein diet, about 1 gm. per kilo body weight per day (the protein in a quart of milk usually suffices to meet the protein requirements), a low salt diet, no condiments or purine diuretics (caffein, urea, etc.), plenty of fluids (at least a third more than that excreted the previous day), mild alkalies, and iron. Warm underclothing to prevent chilling is important. Rest in bed until the urine is negative for at least a week.

Sinuses can be washed out and infected teeth may be removed during the acute stage of the nephritis. In the case of suppurating ears with possible mastoid involvement surgery may be necessary for a cure during the height of the nephritis. Rarely is tonsillectomy indicated during the acute stage.

Case VI. Chronic Diffuse Nephritis.—Unfortunately this patient died about a month ago, but the case is so characteristic

of the most frequent type of chronic nephritis in children that we will review it here and also present the necropsy findings.

The patient, a girl, was attended by both of us at times from infancy. She had a hemorrhage from the bowel shortly after birth which was controlled by intramuscular blood transfusions. During her infancy she was a rather poor eater and had several attacks of bronchitis. The mother had an incipient pulmonary tuberculosis about a year before this pregnancy, but it had healed by the time the patient was born. This was her first and only child. The child developed into a perfectly normal healthy little girl, well-nourished and developed. She had none of the contagious diseases, but was subject to occasional attacks of tonsillitis.

The onset of the nephritis was acute and resembled very much the condition described in Case V. Following a rather severe attack of tonsillitis and cervical adenitis when three years of age, the mother noticed the scanty dark reddish urine, puffiness of the eyelids and face, and a sallow complexion. The physical examination, and blood and urine examinations practically duplicated those described in Case V.

After the usual treatment for acute hemorrhagic nephritis including rest in bed for two months, the condition of the child had improved to the extent that the urine showed albumin + to ++, few microscopic red cells (although grossly clear and yellow) which at times were absent entirely, occasional granular casts, and a few leukocytes. Three months after the onset the child was allowed to be out of bed moderately, but was kept on a low protein and low salt diet with mild alkaline waters and iron therapy.

Four months after the onset an acute exacerbation recurred, more severe than the original attack, with much blood in the urine. However, the patient was not prostrated or apparently acutely ill, and no more edema appeared other than the puffiness about the eyelids and slight pitting about the ankles. Considerable secondary anemia was present, the hemoglobin being 60 per cent., and the erythrocytes, 3,500,000; leukocytes 15,000; 65 per cent. polymorphonuclears. The blood-pressure was 115

systolic and 65 diastolic, the blood-urea 30 mg. per 100 c.c. of blood. Because of the extensive hematuria, intramuscular blood tranfusions were given (30 c.c. every other day for three injections) which diminished considerably the hematuria. Because of the history of intestinal hemorrhage shortly after birth, the coagulation time, bleeding time, and blood-platelet count were carefully investigated. They were all within normal limits, so the patient did not suffer from any hemorrhagic dyscrasia. Because of the history of tuberculosis in the mother, urologic examination was resorted to, but with negative findings as far as tuberculosis, calculus, or tumor of the urinary tract were concerned. The pathologic urine was coming from both kidneys. Guinea-pig inoculations were negative.

The urinary findings improved during the next month, but then remained about constantly as follows: albumin ++, erythrocytes enough to cause a slightly smoky urine, and a few casts. The patient at no time appeared acutely ill, ate well, and seemed happy.

One year after the onset, because her condition was seemingly stationary, tonsillectomy and adenoidectomy were performed under general anesthesia, although the blood-pressure was 120 mg. systolic and 70 diastolic and the blood-urea 20 mg. Shortly following the operation the blood disappeared from the urine, but the albumin remained + to ++ with moderate numbers of pus-cells. The patient was pale and pasty in appearance, but no obvious edema was present.

From time to time about every four months acute exacerbations would occur, usually following a mild upper respiratory infection (no nasal sinus infection was present). These attacks were accompanied by vomiting, abdominal pain, malaise, anorexia, slight fever, bloody urine, and increase in the puffiness about the face. They lasted from two to four weeks. During these attacks the blood-pressure increased to about 130 systolic and 85 diastolic, the blood-urea rose at times to 60 mg., and the secondary anemia became more marked. Between attacks her condition improved considerably, the urine clearing except for ++ albumin and the anemia lessening. Gradually, however,

the blood-pressure remained increased between exacerbations, 140 systolic and 80 diastolic, the blood-urea remained high, and the heart, especially the left ventricle, hypertrophied.

Four years after the onset of the nephritis, about one and a half months ago during an acute exacerbation, the vomiting and abdominal pain became very pronounced, severe headache, dyspnea developed, the odor of the breath was strongly uriniferous, the urine decreased in quantity, the patient appeared acutely ill, but no apparent edema developed. The patient still appeared well nourished, but was pale and waxy looking. The blood-pressure was now 140, the blood-urea 65, the creatinin 4.5 mg. The hypertrophied heart was beginning to decompensate and dilate. Within a week the patient collapsed into coma, developed convulsions, and died a uremic death.

The pathology as reported by Dr. Benson Bloom of Nelson Morris Research Institute was as follows:

At autopsy there was found ascites and edema of the subcutaneous and abdominal tissues and of the feet. The heart was markedly dilated and its walls hypertrophied. The heart weighed 193 grams. In the aorta there were a few raised yellow arteriosclerotic patches which became more marked as the aorta was traced toward its bifurcation. The kidneys were decreased in size, the right one weighing 50 grams, the left, 60 grams. The two kidneys were quite similar. The capsule stripped with some difficulty, although without tearing the renal parenchyma. The surfaces of the kidneys were pale, salmon-brown color, and studded with numerous minute, slightly raised, gray nodules. In addition, there were a number of small petechial hemorrhages, the largest about 2 mm. in diameter. On the cut surface the cortex appeared markedly reduced in size, measuring on an average 3 mm. In it as well as in the medulla there were a number of petechial hemorrhages like those seen on the surface. Throughout the cortex were scattered many minute, orange-yellow patches in addition to pin-point sized glistening plaques. The demarcation between medulla and cortex was not as clear as one usually sees and the striations of the cortex were rather indistinct. The medulla was opaque, gray in color, and in it, too, the striations

were indistinct. About the pelvis there was an increased amount of fat.

In the microscopic examination of the kidneys extreme changes were found involving all the elements of the renal parenchyma. Large numbers of glomeruli were completely obliterated by fibrous tissue proliferation. In others various stages of fibrosis were seen and no normal glomeruli were to be found. Glomerular fibrosis seemed to be mostly within the tufts themselves, although the capsular fibrosis was quite striking. Apparently because of the contraction of the interstitial tissues the number of glomeruli seen in one low-power microscopic field was greatly increased. A number of the more normal glomeruli appeared enlarged, most of the enlargement apparently being due to dilatation of the capsular space. In addition to the fibrotic changes in the glomeruli there were a few glomeruli in which there were collections of leukocytes in the capsular space, and in some there was a suggestion of crescent formation. No actual blood was seen in the capsular space, but in a few of the glomeruli there was a slight amount of fluid. Throughout the rest of the renal parenchyma there was a striking fibrosis and diffuse lymphocytic infiltration with almost complete destruction of the tubular elements. Those tubules that remained were arranged in small groups and appeared to be anatomically associated with the more normal glomeruli. Even in these small collections of tubules there were changes present, consisting of moderate dilatation of the tubules with flattening of the epithelium and fibrosis of the interstitial tissue. In a number of the tubules leukocytes were found and many others contained fresh red blood-cells. Other tubules contained pink staining material. In some there were fully formed hyaline casts. The smaller arteries showed a mild intimal thickening. The collecting tubules in the medulla all appeared dilated, the interstitial tissues fibrosed, and lightly infiltrated with lymphocytes. Many of these tubules contained blood and hyaline casts. Under the thickened renal capsule there were a number of focal lymphocyte accumulations.

In the heart there was seen a diffuse, fine increase of connective tissue in the myocardium, most of which seemed to be in the

neighborhood of smaller blood-vessels. There were no large areas of fibrosis. A section taken through a plaque in the aorta showed a striking intimal sclerosis with deposition of a large mass of fatty tissue in the base of the intima, and in the center of this mass a deposit of granular, calcified material. The intima in other areas was rich in fibrous tissue cells and contained scatterings of lymphocytes.

Chronic nephritis is an infrequent condition in childhood compared with acute nephritis, and is usually seen as a continuation of an acute hemorrhagic nephritis. The progress is irregular and marked by periods of exacerbations and remissions. The exacerbations are due to recurring infections from some source, each one damaging the kidney further, until finally the kidney decompensates beyond redemption much as the chronic recurring endocarditis hearts do. In chronic nephritis following acute hemorrhagic types the major portion of the damage is primarily to the glomeruli, but all the structures of the kidney necessarily suffer to some extent, so that pathologically we are dealing with a diffuse nephritis. The connective tissue gradually replaces the damaged and destroyed glomeruli and increases in the interstitial tissue usually preceded by small round-cell infiltration until in the late stages we get a secondarily contracted kidney much as is seen in the adult. A much rarer form of chronic nephritis in children begins with an infiltration and proliferation of connective tissue in the interstitial regions resulting clinically in a picture of chronic interstitial nephritis resembling the adult type of so-called chronic Bright's disease.

When the glomeruli are mostly involved with little tubular disturbance there is said to be little or no *edema*, but in the case here described even with secondary atrophy and destruction of the tubules (very few tubules left) there was never a marked edema. When edema is slight only the face and ankles are involved. *Anemia* is a prominent symptom. The combination presents a pale, puffy pasty patient. The urine picture varies, although albumin in some amount is always present. During exacerbations the urine is decreased, the albumin increased, and blood, casts, and leukocytes appear. The heart hypertrophies,

the blood-pressure increases, the non-protein nitrogen waste products increase, and death results eventually from acidosis, uremia, or perhaps acute cardiac decompensation.

The treatment consists in retarding the progress of the disease as much as possible and relieving the symptoms as they arise. As exacerbations are usually due to reinfection, this should be avoided by removing all foci of infection when conditions permit, and avoiding contact with other infected children, avoiding exposure to cold by wearing warm clothes, especially woolen underwear, and the removal to a warm even climate during the winter months. Very moderate regular exercise, never carried to the point of fatigue, and as much rest as possible is advisable. The protein and salt in the diet should be moderately restricted. Iron in some bland form as in ferri et ammonii citratis or as Basham's mixture aids the anemia.

During acute exacerbations the same treatment is instituted as described in Acute Hemorrhagic Nephritis.

The retention of the non-protein nitrogenous products in the blood has a somewhat different significance than in adults. The child's kidney apparently is more plastic and can more easily compensate for damaged portions. During the early stages of acute glomerular nephritis these elements are usually moderately increased in the blood, but in chronic nephritis a considerable retention of non-protein nitrogen occurs only in a very severely damaged kidney, and occurs much later in the course of the disease than in adults. A progressive increase of these elements in a child's blood indicates a rather rapidly fatal outcome.

CLINIC OF DR. NATHAN SMITH DAVIS, III

ALEXIAN BROTHERS HOSPITAL

CIRRHOSIS OF THE LIVER

DURING the past few months there have been admitted to my service a series of cases in which there was icterus or enlargement of the liver or congestion in the portal circulation alone or in combination. In some of these cases the diagnosis was relatively simple, but in others it has not been so easy.

Case I.—G. M., a white garage operator, aged fifty-five, was admitted December 11th complaining of pain in the chest, head and abdomen, and of vomiting. These symptoms had commenced about six months before admission, and, in addition, there had been some loss of weight and a slight icterus. The condition of the patient had steadily been getting worse and he was very weak. Up to the onset of the trouble he had been a heavy drinker of whisky, but had stopped drinking when he began to feel ill. During this time he had developed quite a cough, which was nonproductive. He was contipated and his stools were clay colored. The past history was negative except that he admitted a syphilitic infection.

On examination, was found a middle-aged rather emaciated white male, whose skin was quite yellow, whose breath was foul, and who appeared very weak and quite sick. The head and scalp were negative. The pupils were equal and reacted to light and accommodation; the sclera were icteric. The teeth were in very bad condition; the pharynx was clear though red; the tonsils were not enlarged. The chest was symmetrical, expansion equal, but breathing was shallow; there were no areas of consolidation,

altered breath sounds, or râles found. The heart was normal in size (left heart dulness 10.5; right heart dulness 3 cm. from midline); there were no murmurs or altered tones. The liver was palpable 2 cm. below the costal margin, its edge was smooth, but very firm. The spleen was palpated at the costal margin on deep inspiration. There was a moderate ascites. The lower intercostal, peri-umbilical, and inferior hemorrhoidal vessels were dilated. The blood-pressure was 100/48. Respirations varied from 20 to 24, the temperature from 98° to 100° F., the pulse rate from 80 to 100. The urine specific gravity was 1012 and it contained albumin and bile. The Wassermann was negative; the icterus index 20; the stools contained occult blood at two examinations; vomitus contained no free acid, and had a total acidity of 30. There was a marked secondary anemia, reds 1,520,000; whites 14,200, and hemoglobin of 20; the differential count was not remarkable, though macrocytosis and poikilocytosis was noted. He was placed on Koessler's anemia diet (high vitamin) and given dilute hydrochloric acid in doses of 2 c.c. well diluted before, during, and after each meal, and 4 c.c. of cod-liver oil in orange juice three times daily. He continued to grow weaker and became somewhat delirious; had severe vomiting with coffee-ground vomitus. He died on December 23d. Unfortunately it was impossible to obtain a necropsy.

In the differential diagnosis of this case three things had to be specially considered—pernicious anemia, malignancy, and cirrhosis of the liver. The color of the skin, the severe anemia, with macrocytosis, though with a low color index, made one think of pernicious anemia at once. However, the enlarged liver and spleen, ascites, dilatation of the vessels in the collateral portal circulation, together with the history of syphilis and alcoholism, made this seem less probable than cirrhosis of the liver. Malignancy would explain the findings readily if it were not for the enlarged spleen and the total absence of any symptoms or signs of trouble in the gastro-intestinal tract such as are usually found in malignancy, that is, obstruction and a tumor mass. So while this was not confirmed by necropsy, it seemed that alcoholic cirrhosis was the most probable diagnosis.

Case II.—B. L., a white janitor, aged fifty-nine, was admitted to the hospital February 19th complaining of swelling of the abdomen of four weeks' duration. He stated that he had felt well up to four weeks before admission, when his abdomen had commenced to swell quite rapidly until it had become very much distended. The only discomfort mentioned was that due to this distention. As the abdomen had enlarged the urinary excretion had diminished until he was passing almost no urine at all; catheterization before admission to the hospital had removed only about 60 c.c. from the urinary bladder. The family and past history were negative except that he admitted a chancre twenty-three years before for which he had had intensive treatment for about two years, and that he had always been a heavy beer drinker.

Physical examination revealed a fairly well-nourished white male who was not acutely ill, but whose abdomen was very noticeably enlarged. The pupils were equal, regular, and reacted to light and accommodation. The chest was symmetrical and expansion equal, though diminished, due to the limitation of diaphragmatic movements by the distended abdomen. No pathology was found in the lungs or heart. The blood-pressure was 128/74. The abdomen was tremendously distended and there was a dulness in the flanks shifting with change of position, which extended almost to the borders of the M. rectus abdominalis when the patient was flat on his back. There was a small easily reducible umbilical hernia. The liver dulness extended from the fifth to the eighth rib. The abdominal wall was so tense that it was impossible to palpate any organs, but there were no areas of tenderness. There was a slight edema of the extremities. The patellar reflexes were present, but diminished. The urine specific gravity was 1026 and it contained no albumin, sugar, casts, or other pathologic elements. The erythrocytes numbered 4,480,000; leukocytes, 8000, and the hemoglobin 85 per cent. The Wassermann was negative and, although there was no visible jaundice, the icterus index was 12.

A paracentesis abdominalis was performed at once and about 16,000 c.c. of clear yellow fluid was removed. After this it was

possible to palpate the abdominal viscera, and the liver was about 1 inch above the costal margin, firm, and with a rough surface; it seemed much more freely movable than normal. The spleen was firm and palpable at the costal margin. The abdomen refilled rapidly, and eight days later a second paracentesis was performed and 12,000 c.c. were removed. As the abdomen began to refill rapidly, it was thought advisable to do an omentopexy fifteen days after admission. This was done by Dr. M. L. Harris, but sufficient time has not elasped to determine its efficacy in this case. At the operation a typical "hob-nail" liver was found so that the preoperative diagnosis of cirrhosis was confirmed.

CARCINOMA OF THE PANCREAS WITH METASTASES TO THE LIVER

Case III.—J. B. B. On December 4, 1926 a watchman aged seventy-three, single, was admitted to the hospital complaining of vomiting, loss of weight, and loss of appetite. About two months prior to admission patient began to vomit shortly after getting up in the morning and before eating breakfast. The vomitus consisted of undigested food eaten the previous evening and was quite sour. There was no visible blood or "coffee-ground" vomitus and the vomiting was not preceded by nausea. In the morning before breakfast while on his way to work the patient would feel inclined to vomit, would "swallow his finger," and throw up a quantity of undigested food. The vomiting was always before breakfast. He had no appetite except before his evening meal, and after it had a feeling of weight in the upper abdomen, and during the night there was often regurgitation of food. For the two months prior to admission he had been losing weight so rapidly that his fellow workers commented on it. He had no pain or discomfort except that cited above.

Previous History.—Rheumatism for nine weeks about 1911. "Fever and ague" as a child. No operations. Venereal diseases denied.

Physical examination reveals an elderly white male whose skin has a lemon-yellow tint and who looks anemic and cachectic, but does not appear acutely ill. The head and scalp were negative. The pupils react to light and accommodation. Many teeth are missing and there has been a good deal of dental work done on those remaining. A small lymph-gland is palpated in the left supraclavicular fossa. The chest is symmetrical, expansion normal and equal, and the breath sounds are normal. The heart is not enlarged and there are no murmurs. The skin over the abdomen is dry and there is little subcutaneous fat. There is a large mass visible in the epigastrium and right hypochondrium.

On palpation, the liver border is felt at the level of the umbilicus and its surface is roughened. It seems to contain a large rounded non-fluctuating mass about 15 cm. in diameter. This is not tender. The spleen is not palpable. On rectal examination nothing pathologic was noted. There was no swelling of the extremities. The reflexes were normal.

The urine specific gravity was 1018, and it contained no albumin, sugar, ketone bodies, casts, blood, or pus. The erythrocytes numbered 4,160,000, the whites 16,200, and the hemoglobin was 60 per cent. The icterus index was 6.2. A forty-minute Ewald had a total acidity of 5 and contained no free acid or lactic acid; a few blood-cells and epithelial cells were found. The stool contained occult blood. On fluoroscopic examination the stomach filled normally, the movements were somewhat sluggish, the duodenal cap was normal in shape, but there seemed to be an obstruction in the first part of the duodenum due to pressure from without.

Under dietary management the symptoms were somewhat relieved and the patient was discharged, only to return in a few days as the symptoms had recurred. Medical management proved of no value and so an exploratory laparotomy was performed by Dr. M. L. Harris to see if any surgical relief were possible. The preoperative diagnosis was carcinoma with metases in the liver and regional lymph-glands. Despite the fact that the stools contained occult blood, it was not thought that the primary tumor was in the intestinal tract. The pancreas or bile passages seemed to be the most probable locations, and the absence of jaundice made one inclined to incriminate the gall-bladder. At operation a tumor of the pancreas was felt which was adherent to the stomach. The diagnosis made was that of carcinoma of the pancreas with involvement of the stomach by extension and with metastases in the liver. The patient recovered from the operation, but, as was expected, has continued to grow weaker and more anemic. He now has a moderate ascites and marked edema of the extremities and is quite irrational much of the time. It is probable that he will live only a very short time.

Case IV.—M. K., a man aged sixty-nine, was admitted to the hospital September 20, 1926 complaining of jaundice and pruritis. This began about seven months prior to admission without pain and with a gradual onset. He has at no time had any attacks of pain and has no abdominal discomfort of any kind. The pruritus is very severe and the jaundice deep. He has had roentgenograms made of the gall-bladder region which were negative. He has lost a good deal of weight in the seven months. From time to time he has had chills and fever.

Family History.—Father died of kidney trouble at sixty-two; mother died at forty-five, cause not known; 1 brother and 2 sisters living and well; one brother died at sixty-one. There is no history of hereditary disease.

Past History.—No previous illnesses of consequence. Herniotomy, right, in 1908, and again in 1923. Denies venereal disease. Nocturia, but no dysuria or other difficulty. He is somewhat constipated and has clay-colored stools.

Physical examination reveals a moderately well-nourished white male, markedly jaundiced, and with multiple excoriations all over the body from constant scratching, who does not appear acutely ill.

Head and scalp negative. Pupils equal and react to light and accommodation. The scleræ are markedly icteric. Nose, ears, mouth, and neck are negative. The chest is symmetrical, expansion normal and equal, breath sounds normal. The heart is not enlarged; there are no murmurs. The abdomen is negative; no tumors palpable and no tenderness anywhere. The liver is not enlarged and the spleen is not palpable. The reflexes are normal.

Urine specific gravity was from 1010 to 1015, and contained a trace of albumin from time to time, and also some granular casts. Bile was always found, but the amount varied greatly from day to day.

Blood.	9/21/26	9/29/26	10/13/26	10/29/26	11/20/26	12/2/26
Reds	2,904,000	..	4,080,000	3,840,000		
Whites	11,000	..	7,200	18,000		
Hb	85	..	80	75		
Poly. N	82	..		70		
S. mono	16	..		28		
L. mono	1	..				
Poly. E	1					
Poly. B						
Trans				2		
Icterus	.....	..				
Index	.....	25	.....	.....	34	25

The stools never contained blood and sometimes no bile, at other times appreciable amounts. Fatty acid crystals were present. After tetraiodophenolphthalein by mouth the gall-bladder was faintly visible; the shadow was symmetrical and uniform in density; there was no evidence of stones.

During his stay in the hospital he gained 4 or 5 pounds in weight, and the jaundice cleared up considerably. The pruritis almost disappeared. He had some furuncles of the scalp which cleared up with autogenous vaccines. The blood-picture improved materially. During this time he ran an irregular temperature; sometimes it would be normal for several days and the icterus would begin to clear and the stools become darker colored. Then he would have chills and a fever which at times reached 103° F., after which the icterus would be more marked. As to treatment, he was given methanamin in 0.30 doses three times a day much of the time, and was on a high fat diet, taking milk and cream with an egg between meals in order to promote emptying of the bile passages.

The differential diagnosis in this case was very difficult. Here was a man nearly seventy, who on admission had been jaundiced for seven months, who was in a fair state of nutrition, and not very anemic and not acutely ill. Examination revealed nothing but the jaundice and the excoriations from the resulting pruritus. Laboratory tests and the Roentgen ray gave little if any additional information. During his stay in the hospital he has gained in weight and strength, but the jaundice has persisted, though it is gradually lessening, and the pruritus has al-

most disappeared. From time to time there have been chills and fever accompanied by an increase in the jaundice, but not associated with pain. In a man of this age with a painless jaundice malignancy is the first thing thought of, but the absence of an enlarged gall-bladder, the frequent presence of bile in the stools, and the general condition of the patient seven to ten months after the onset make this seem highly improbable. The complete absence of pain and negative roentgenological findings make cholelithiasis with a ball-valve stone in the common duct seem to be an unlikely diagnosis. The absence of any palpable change in the liver or of any changes in the portal collateral circulation and the absence of ascites practically rule out cirrhosis of the liver. There remains, then, chronic catarrhal or infectious jaundice which will account for all the symptoms and findings and the improvement under a high fat diet plus methenamin, remains as the most probable diagnosis. While it is questionable just how much good a cholecystotomy would have done if this is the correct diagnosis, it is believed that such an operation was indicated if only to rule out the possibility of common duct stone or non-malignant pressure from without as the cause of the jaundice. The patient, however, steadfastly refused operation even though he did think that improvement was very slow.

Case V.—R. C., a white mechanic, aged sixty-five, entered the hospital November 4th, complaining of cramp-like pains in both legs on exertion; that his left leg felt as if it were "wrapped with a bandage of lead"; that the joints of his fingers were swollen, and that the hands were practically useless due to the stiffness and pain; that there were small cream-colored areas in the skin of the left index-finger, terminal phalanx, and a large firm painless nodule on the outer surface of the right heel that contained material similar in appearance. The onset of the above symptoms had been about one year previous to admission and they had gradually been getting worse, so that for several months he had been unable to work.

On further questioning he stated that about two and a half years before admission he had first noticed a peculiar reddish-purple discoloration of the nose and hands, and that the vessels of the nose had become more prominent. He was very constipated, and had noticed a tender mass in the left upper quadrant a few months prior to admission.

His past history was negative except that he had been a heavy drinker before prohibition, and that a few months ago he had had a severe hemorrhage following the extraction of some teeth. There was no history of any similar disease in any of his relatives or was there any evidence of other hereditary disease in the family.

Physical examination revealed an elderly white man, fairly well nourished, whose skin had a peculiar reddish cyanosis and who seemed weak and quite uncomfortable, though not acutely ill. The conjunctivæ were hyperemic; the pupils equal and reacted to light and accommodation; the nose was hyperemic and the reddish cyanosis was quite marked. There were many carious teeth. No pathology was found in the lungs. The heart was slightly enlarged, 11.5 cm. to left of midsternal line, but the sounds

were clear and there were no murmurs. The blood-pressure was 120/70. The spleen is enlarged and extends down to the level of the umbilicus and is slightly tender. The liver is also enlarged and extends 5 cm. below the costal margin. Otherwise the abdomen is negative. There is no ascites. The joints of the fingers are swollen and there are cream-colored areas in the skin of the left index-finger about 1 mm. in diameter. On the outer surface of the right heel there is a firm, hard, non-fluctuating tumor containing some yellowish material from which the patient states "pus" had been removed. This nodule is not tender or painful.

The admission specimen of urine had a specific gravity of 1018 and contained no albumin, sugar, ketone bodies, casts, blood- or pus-cells. Blood-counts have been as shown on page 227.

On November 6, 1926 the blood uric acid was 4.3 and on December 7, 1926 the icterus index was 10. All stools examined were negative for occult blood.

Ophthalmologic examination by Dr. Oscar H. Kraft on December 3, 1926 revealed the following: "Patient states his vision to be very good for distance; near vision is corrected by glasses. Ophthalmoscopic, both eyes negative; vessels and retina normal; lens and vitreous clear; the disk is slightly darker red than usual, but not pathologic.

"On December 13th ophthalmoscopic examination showed disks not quite as dark red as ten days ago, otherwise the same. Vessels may be slightly contracted, but would call them normal."

It was quite evident that the patient had podagra with uric acid tophi in the skin of the finger and on the right heel. This diagnosis would, however, not explain the enlarged liver and spleen. He was at once put on a purin poor diet and given cincophen in doses of 0.45 gm. four times daily. The fingers and other joints began to improve almost at once and have continued to do so ever since, though there have been one or two slight exacerbations since. Roentgenograms confirmed the diagnosis of podagra.

It was more difficult to explain the enlarged spleen and liver until the second blood-count was made, which made the diagnosis of erythremia clear. Roentgenotherapy over the long bones was

	11/5/26	11/9/26	11/15/26	11/18/26	11/20/26	11/22/26	11/24/26	11/26/26
Reds	5,536,000	8,220,000	8,960,000	8,320,000	8,160,000	8,320,000	8,120,000	8,720,000
Whites	11,600	17,600	13,200	15,200	12,600	20,800	13,400	10,800
Hb	106	115	120	120	120	120	125	125
Poly. N	82	83	...	...	...	72		
S. mono	10	9	...	...	...	20		
L. mono	1	2						
Poly. E	4	6	...	...	...	4		
Poly. B	...	...	...	...	...	2		
Trans	3	...	...	...	...	2		

	11/29/26	12/1/26	12/3/26	12/7/26	12/10/26	12/13/26	12/20/26	12/22/26
Reds	8,400,000	7,920,000	7,840,000	8,160,000	7,920,000	8,080,000	8,320,000	8,240,000
Whites	13,000	13,200	13,200	14,200	17,600	11,400	13,600	13,600
Hb	125	120	120	120	115	115	120	120

	12/24/26	12/27/26	12/29/26	1/3/27	1/5/27	1/7/27	1/12/27	1/15/27
Reds	7,680,000	8,960,000	7,840,000	8,080,000	8,240,000	8,320,000	8,400,000	7,680,000
Whites	19,000	18,000	11,400	9,400	10,000	14,000	10,000	12,200
Hb	120	120	120	120	120	125	125	120

	1/17/27	1/26/27	1/31/27	2/4/27	2/7/27
Reds	8,512,000	6,080,000	8,160,000	7,680,000	8,080,000
Whites	16,000	11,000	12,000	10,000	9,400
Hb	120	120	120	115	120
Poly. N	...	...	...	83	...
S. mono	...	...	...	10	...
Poly. E	...	...	...	5	...
Poly. B	...	...	...	1	...
Trans	...	...	...	1	...

started at once and benzol was administered in doses varying from 0.18 to 0.60 c.c. three times daily. As after two courses of roentgenotherapy and two months on benzol there had been no decided change in the blood-count, phenylhydrazin hydrochlorid in 0.1-gm. doses daily was instituted. By this time the podagra had improved so that the cincophen was cut to one dose a day. After one month of the phenylhydrazin and a third series of roentgenotherapeutic treatments over the long bones there is still a high blood-count, but the general condition of the patient is very much improved. It is planned to increase the dosage of phenylhydrazin to 2 capsules daily and also give more intensive roentgenotherapy in order to get the blood-count down to within normal limits.

CLINIC OF DR. ARTHUR R. ELLIOTT

MEDICAL SERVICE, ST. LUKE'S HOSPITAL

TYPHOID BONE INFECTION

T. D., white male aged thirty-six, entered St. Luke's Hospital complaining of pain in the left hip and groin of two weeks' duration.

The personal history related the fact that when twenty-five he had a severe attack of typhoid fever lasting with relapses about six months in all. After the height of the fever had passed he developed pain in the left leg and both knees became swollen and painful. When convalescence was complete he found that he could not use his left hip freely and he has walked with a slight left-sided limp ever since. He had no abscesses either of bones or glands, and no gall-bladder complication. He has never been known to be a "carrier." Ten years after the initial typhoid infection he again developed acute arthritis of the left knee-joint. This mono-arthritis confined him to bed for two weeks, after which it rapidly subsided without any resulting deformity or disability of the joint. He returned to his work after a total loss of only four weeks' time. He very definitely states that at no time during this illness was he conscious of pain or soreness in the left hip.

The history of the present illness is as follows: Two weeks before admission, while moving a heavily loaded truck, he slipped on the wet floor and in an effort to recover his balance twisted his body sharply. Immediately a sharp, but transitory pain shot through his left hip region. He resumed his work, but within a few hours the pain returned severely and persisted, confining him thereafter to bed. He had some fever, but had not himself noticed any swelling about the seat of pain. Examination re-

vealed a firm rounded mass in the lower left quadrant with its long diameter oblique and parallel to Poupart's ligament and about 2 inches above it. Its inner edge was about at the parasternal line and the saphenous ring. The mass was firmly fixed and decidedly tender to pressure. There was also some tenderness to pressure over the anterior superior spine of the ilium. The genitalia appeared normal and no evidence of hernia existed. There was some muscular rigidity in the left flank and lower abdominal quadrant. Apart from this the abdomen was without tension or tympany and the liver and spleen appeared normal in size. There was definite limitation of movement of the left hip-joint in all directions, especially in abduction, both active and passive motions producing pain. There was no evidence of capsular distention or irritation posteriorly or laterally. Digital rectal examination revealed nothing significant. The urine sediment contained a few erythrocytes and leukocytes. Urine cultures showed no growth. There was a leukocytosis of 24,700. A septic type of fever was present with afternoon elevation of 2° to 4° F. The Widal test showed positive agglutination with Bacillus typhosus. Roentgenograms of the left hip showed the joint space to be very indistinct as though an exudate existed. The articular surfaces could be distinctly outlined. There was evidence of new bone formation involving the upper half of the head of the femur. A diagnosis was made of a suppurative bone lesion possibly of remote typhoid origin.

Blood-cultures proved negative and neither the urine nor feces (several specimens) yielded Bacillus typhosus. The left leg was placed in extension. The leukocyte count continued high, with marked afternoon flights of temperature and morning remissions to near normal. Pain persisted with slow increase in the size of the swelling. Three weeks following admission fluctuation could be made out and pus was aspirated. From this material Bacillus typhosus was grown in pure culture. One week later the mass ruptured spontaneously, discharging a quantity of thin purulent fluid. A few days later a second fluctuant mass developed in the soft tissues of the left thigh just distal to Poupart's ligament, and this was opened under local anesthesia by

Dr. Fisk, about 2 ounces of thin pus being evacuated. From this pus Bacillus typhosus was grown in pure culture. Both suppurating tracts were later injected with lipiodol solution and roentgenograms made of the region (Fig. 19). These revealed the head of the femur to be flattened and thickened and the joint space narrow. There existed a good deal of new bone formation

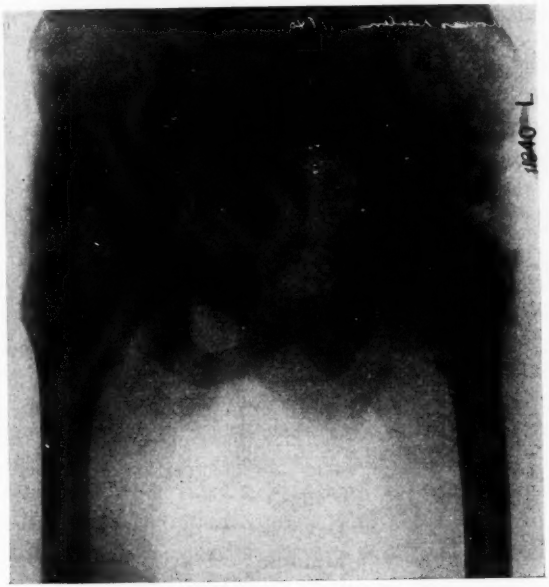

Fig. 19.—Roentgenogram showing new bone formation about head of left femur and multiple sinuses extending into soft tissues above and below joint.

extending outward into the soft tissues from the superior and inferior borders of the head of the femur. There appeared multiple sinuses outlined by the iodid solution extending upward to the anterior superior spine of the ilium and downward toward the head of the femur. Progress thenceforward was uneventful, patient being discharged with wound healed after a hospital residence of seventy-six days.

Laboratory data:

Widal tests

Patient's serum against known strains:

Strain R ⟶ + up to 1 : 100
Strain G ⟶ + up to 1 : 150
Para A ⟶ 0
Para B ⟶ 0

Repeated:

Strain R ⟶ + up to 1 : 320
Strain G ⟶ + up to 1 : 640
Para A ⟶ 0
Para B ⟶ + up to 1 : 80

Patient's organism against known serum:

⟶ positive up to 1 : 3000

Pus from abscess:

Direct stain (Gram) many leukocytes, no bacteria.

Cultures:

Blood agar ⟶ mixed growth transferred to
Endos. ⟶ non-lactose splitting Gram-negative bacilli
Plain agar ⟶ pure culture Gram-negative bacilli
Broth ⟶ motility (hanging drop)
Milk ⟶ acid—no coagulation for twenty-one days
Sugars ⟶ Dextrose ⟶ acid; Sacch. ⟶ 0; Lact. ⟶ 0; Raff ⟶ 0; Salicin ⟶ 0 } = Bac. Typhosus.
Marmite ⟶ acid
Xylose ⟶ acid

Guinea-pig inoculations:

One yellow pig, 1 c.c. of pus injected subcutaneously ⟶ abscess locally which broke and discharged yellow thick pus. When killed thirty days later no tubercles found in liver, spleen, or lymph-nodes. Pus from abscess cultured and Bacillus typhosus isolated.

Discussion.—This case is an interesting and illustrative instance of typhoid bone infection, latent for many years, and finally

becoming active under the influence of indirect trauma. The interval period elapsing between the initial attack of typhoid fever and bony complications in our case is eleven years. Although in the majority of cases this complication occurs during the course of the fever or while the patient is convalescing, its appearance may be postponed for months or even many years. Von Bisselick has reported periostitis of a rib the pus from which gave a pure culture of the bacillus eighteen years after the typhoid attack, and Keith and Keith in a recent publication mention its occurrence as late as forty-five years. In this statement they probably have in mind the case reported by Buschke, in which forty-six years had elapsed since the original fever, yet the typhoid bacillus found in the pus was capable of further growth and cultivation. My own experience includes a case of typhoid osteomyelitis following an injury, the pus containing Bacillus typhosus twenty-five years after the initial infection.

The frequency of typhoid bone infection is difficult to compute, since in many cases the bacteriology is not determined. Miller, in Oxford Medicine, estimates that 1 per cent. is a fair average. Keith and Keith state that its frequency as given by fifteen authors is 164 times in 18,840 cases (0.87 per cent. of all cases having metastatic infections in the bone). In 700 cases of osteomyelitis only 3 were due to typhoid (0.43 per cent. of all osteomyelitis). Ever since inoculation experiments with the typhoid organism have been studied it has been apparent that the bone-marrow like other lymphoid structures of the body is a favorable location for the bacillus, and it may continue to multiply here long after the general infection has subsided and the temperature returned to normal. It is consequently not astonishing that infection of the bones is a not uncommon sequela of typhoid fever. The long bones of the extremities are the most frequently involved, although other bones, such as the sternum, ribs, and clavicle, may be affected. The lesion is most frequently periostitis, although osteomyelitis and osteitis may result. When an abscess forms and is drained, examination of the pus may lead to identification of its etiology. Other cases pursuing a non-suppurative course develop slowly, with little systemic reaction,

vealed a firm rounded mass in the lower left quadrant with its long diameter oblique and parallel to Poupart's ligament and about 2 inches above it. Its inner edge was about at the parasternal line and the saphenous ring. The mass was firmly fixed and decidedly tender to pressure. There was also some tenderness to pressure over the anterior superior spine of the ilium. The genitalia appeared normal and no evidence of hernia existed. There was some muscular rigidity in the left flank and lower abdominal quadrant. Apart from this the abdomen was without tension or tympany and the liver and spleen appeared normal in size. There was definite limitation of movement of the left hip-joint in all directions, especially in abduction, both active and passive motions producing pain. There was no evidence of capsular distention or irritation posteriorly or laterally. Digital rectal examination revealed nothing significant. The urine sediment contained a few erythrocytes and leukocytes. Urine cultures showed no growth. There was a leukocytosis of 24,700. A septic type of fever was present with afternoon elevation of 2° to 4° F. The Widal test showed positive agglutination with Bacillus typhosus. Roentgenograms of the left hip showed the joint space to be very indistinct as though an exudate existed. The articular surfaces could be distinctly outlined. There was evidence of new bone formation involving the upper half of the head of the femur. A diagnosis was made of a suppurative bone lesion possibly of remote typhoid origin.

Blood-cultures proved negative and neither the urine nor feces (several specimens) yielded Bacillus typhosus. The left leg was placed in extension. The leukocyte count continued high, with marked afternoon flights of temperature and morning remissions to near normal. Pain persisted with slow increase in the size of the swelling. Three weeks following admission fluctuation could be made out and pus was aspirated. From this material Bacillus typhosus was grown in pure culture. One week later the mass ruptured spontaneously, discharging a quantity of thin purulent fluid. A few days later a second fluctuant mass developed in the soft tissues of the left thigh just distal to Poupart's ligament, and this was opened under local anesthesia by

Dr. Fisk, about 2 ounces of thin pus being evacuated. From this pus Bacillus typhosus was grown in pure culture. Both suppurating tracts were later injected with lipiodol solution and roentgenograms made of the region (Fig. 19). These revealed the head of the femur to be flattened and thickened and the joint space narrow. There existed a good deal of new bone formation

Fig. 19.—Roentgenogram showing new bone formation about head of left femur and multiple sinuses extending into soft tissues above and below joint.

extending outward into the soft tissues from the superior and inferior borders of the head of the femur. There appeared multiple sinuses outlined by the iodid solution extending upward to the anterior superior spine of the ilium and downward toward the head of the femur. Progress thenceforward was uneventful, patient being discharged with wound healed after a hospital residence of seventy-six days.

Laboratory data:

Widal tests

Patient's serum against known strains:

Strain R ⟶ + up to 1 : 100
Strain G ⟶ + up to 1 : 150
Para A ⟶ 0
Para B ⟶ 0

Repeated:

Strain R ⟶ + up to 1 : 320
Strain G ⟶ + up to 1 : 640
Para A ⟶ 0
Para B ⟶ + up to 1 : 80

Patient's organism against known serum:

⟶ positive up to 1 : 3000

Pus from abscess:

Direct stain (Gram) many leukocytes, no bacteria.

Cultures:

Blood agar ⟶ mixed growth transferred to
Endos. ⟶ non-lactose splitting Gram-negative bacilli
Plain agar ⟶ pure culture Gram-negative bacilli
Broth ⟶ motility (hanging drop)
Milk ⟶ acid—no coagulation for twenty-one days
Sugars ⟶ Dextrose ⟶ acid; Sacch. ⟶ 0
Marmite ⟶ acid; Lact. ⟶ 0
Xylose ⟶ acid; Raff ⟶ 0
Sali-cin ⟶ 0
} = Bac. Typhosus.

Guinea-pig inoculations:

One yellow pig, 1 c.c. of pus injected subcutaneously ⟶ abscess locally which broke and discharged yellow thick pus. When killed thirty days later no tubercles found in liver, spleen, or lymph-nodes. Pus from abscess cultured and Bacillus typhosus isolated.

Discussion.—This case is an interesting and illustrative instance of typhoid bone infection, latent for many years, and finally

becoming active under the influence of indirect trauma. The interval period elapsing between the initial attack of typhoid fever and bony complications in our case is eleven years. Although in the majority of cases this complication occurs during the course of the fever or while the patient is convalescing, its appearance may be postponed for months or even many years. Von Bisselick has reported periostitis of a rib the pus from which gave a pure culture of the bacillus eighteen years after the typhoid attack, and Keith and Keith in a recent publication mention its occurrence as late as forty-five years. In this statement they probably have in mind the case reported by Buschke, in which forty-six years had elapsed since the original fever, yet the typhoid bacillus found in the pus was capable of further growth and cultivation. My own experience includes a case of typhoid osteomyelitis following an injury, the pus containing Bacillus typhosus twenty-five years after the initial infection.

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and gradually subsiding, result finally in local thickening and deformity of the bone—a hypertrophic osteitis. There may be nothing to identify the true character of this bony reaction except perhaps a positive Widal test.

A consideration of the facts revealed in the history of this interesting case leads to the following clinical argument: Eleven years prior to abscess formation there occurred a prolonged and stormy attack of typhoid fever. During the course of this fever there developed pain in the left leg and the knee-joints became tender and swollen. After convalescence was established motion in the left hip was found to be restricted so that the patient walked with a permanent limp. In retrospect it seems apparent that osteitis of the head of the femur from localization of the typhoid organism had occurred. The arthritis of the knee-joints spontaneously subsided without injury to their structure or function. Joint involvement in typhoid fever is an uncommon complication. It may constitute a simple toxic joint reaction, such as is apt to occur during any severe systemic infection, rapidly disappearing as the parent infection subsides. Or, more rarely, it may result from extension into the joint structures of an osteitis in the neighboring long bone. Such an osteo-arthritis will be more severe and prolonged and leave behind some permanent joint disability. In view of the fact that the arthritis in this case spontaneously subsided and disappeared during convalescence, leaving behind no ill-effects, I think we may conclude that it was simple toxic arthritis. If this is accepted as a fair assumption, it then becomes likely that the second arthritis noted in the history as occurring one year ago and ten years after the typhoid fever was likewise a simple arthritis, but whether produced by an occult lighting up of the latent typhoid infection in the femur producing a specific toxic reaction in the joint or due perhaps to some other and incidental infective factor must remain an open question. The fact that it occurred only in the knee of the left and infected leg is, of course, a suspicious circumstance, raising a certain presumption in favor of its specific origin, but this may be mere coincidence. The matter cannot be settled by argument and must be left undecided. We may at

least consider it certain that the knee did not become involved from extension of typhoid osteitis into the knee-joint.

Summary.—After a latency of eleven years a typhoid osteitis of the femur becomes active under the stimulation of trauma, forming multiple sinuses in the pus from which Bacillus typhosus is cultivated. When injected subcutaneously into a guinea-pig an abscess results, the pus from which when cultured yields Bacillus typhosus.

PELLAGRA SECONDARY TO CARCINOMA OF THE COLON

E. C., married white woman, entered St. Luke's Hospital April 24, 1925 in a much emaciated condition complaining of sore mouth and diarrhea.

The clinical history records that in 1914 she had undergone hysterectomy for what proved to be on section carcinoma of the uterus. In 1916 she was again operated upon, resection of a portion of the transverse colon being done for a new growth which was reported by the pathologist to be cylindrical cell carcinoma. Following this operation she regained her normal weight and strength and was free from abdominal symptoms until January, 1924, when she began to experience abdominal pains and loss in weight. On examination a mass could be plainly felt in the right lower abdominal quadrant and blood was found present in the stools.

Roentgen examination with barium enema revealed a constant irregular filling defect in the region of the palpable mass. Here for several inches involvement of the colon was apparent indicating an infiltrating growth having the appearance of a carcinoma. She was given five deep *x*-ray treatments between the dates May 29th and June 4, 1924. On the latter date blood-count was 3,475,000; leukocytes, 12,600; and hemoglobin, 60 per cent. No further observations of the patient's clinical progress are available until April 24, 1925, when she entered St. Luke's Hospital on the medical service complaining of emaciation, extreme weakness, vertigo, sore mouth and lips, abdominal cramps, persistent diarrhea, and a skin eruption involving the hands, forearms, feet, legs, and less conspicuously other areas of the skin surface. The account given was that about four months prior to admission she had developed diarrhea which proved intractable to ordinary treatment. In attempting to control this symptom she had reduced food intake to the lowest proportions, confining her diet for many weeks to cereal gruels,

crackers, and tea. Despite this extreme abstinence the diarrhea continued unabated, bowel movements numbering six to twelve daily, often with blood and mucus in the stool. Under this depletion the body weight had become reduced from 115 to 70 pounds. About a month previous to entry an erythematous patch had appeared on the dorsum of each hand, gradually spreading up the forearms, with some local discomfort and itch-

Fig. 20.

ing. Subsequently the dorsum of each foot and later the legs became similarly affected. Coincidently the mouth and tongue became sore, adding to the difficulties of alimentation. The skin and mouth conditions progressed during the next three weeks, but their true nature was not recognized until she was examined at the hospital. It was found that the skin lesions were symmetric with typical pellagrous distribution (Figs. 20–26). They

involved the entire dorsal surface of both hands and continuously the dorsal area of both radial surfaces of the forearms, and also the skin covering both outer condyles. There was a patch about the size of the palm of one's hand on the back of the neck, another one above the episternal notch, and a small one as large as a silver dollar in the right sternoclavicular area. There existed

Fig. 21.

Fig. 22.

a less well-developed but distinctly noticeable involvement on the face, appearing as a scaly patch of butterfly configuration below each inner canthus and along the nasal margins. Both feet on their upper surfaces and the lower half of both legs displayed the most extreme skin changes. All affected skin areas were well demarcated, slightly raised, thickened, deeply pigmented, and scaling; except the dorsum of the right foot, which appeared

raw and moist. The entire mucous membrane of the mouth and tongue had a dull red, beefy, dry appearance. Small denuded areas existed on the tongue, but there was no ulceration. The lips were dry and cracked. Much buccal discomfort was complained of. There was very little discomfort from the hands and feet so long as they were protected from friction. The patient

Fig. 23.

Fig. 24.

was normally oriented, bright, and intelligent. The diarrhea which persisted with from sixteen to twenty-four stools daily was accompanied with cramp-like pains. The stools contained blood and mucus and were uniformly negative to search for ameba and other parasites. Blood examination revealed 2,230,000 erythrocytes, 7250 leukocytes, and 30 per cent. hemoglobin. Blood-cultures and blood Wassermann were negative. There

was a moderate afternoon rise of temperature. Abdominal examination showed a moderately tympanitic and diffusely tender belly, with no signs of free fluid and no visible peristalsis. In the right lower quadrant was an easily palpable mass about the size of a hen's egg, irregular and nodular in outline and firmly fixed. The liver was slightly enlarged and smooth in outline,

Fig. 25.

Fig. 26.

spleen not palpable. No enlarged glands could be felt. General physical examination revealed nothing significant.

The diagnosis of pellagra of starvation type was made and the patient put on a high caloric, high protein diet, containing scraped fresh beef, milk, cream, eggs, puréed peas, orange, and grape-fruit juices.

Following the institution of this régimen a slow but steady improvement in the skin condition occurred, and by June 1st, five weeks after the diet was changed, the last of the skin lesions had disappeared. There was no recurrence during the balance of life. Improvement in the diarrhea was less conspicuous, the number of movements being reduced only to four to six daily. On September 14th barium was administered by mouth and a roentgenogram made. The cecum was very irregular in outline and there appeared to be a constriction at the juncture with the ascending colon. The *x*-ray plate showed an area in the right half of the body of last lumbar vertebra having the appearance of a metastasis. On September 9th the blood-count showed 1,580,000 erythrocytes, and 14 per cent. hemoglobin. Progressive exhaustion ended in the patient's death January 6, 1926. No autopsy was obtained.

Discussion.—Pellagra secondary to disease of the gastro-intestinal tract of such nature as to seriously interfere with nutrition has recently elicited attention. Since 1919 five reports have appeared in the literature. The rarity of mention of pellagra in conjunction with gastro-intestinal disease and the almost complete failure hitherto to report cases of the type suggests that the association has been overlooked or disregarded. Ralph, in the Canadian Medical Journal for April, 1919, describes a case in which skin and mucous membrane lesions typical of pellagra appeared four months before death in a patient who was demonstrated at autopsy to have carcinoma of the gastric cardia. The cardiac stoma was involved in such manner as to considerably interfere with the passage of food from the esophagus. There was increasing dysphagia, regurgitation, and emaciation. Stimulated by this report, Bryan in Virginia Medical Monthly for August, 1919, described an instance of starvation pellagra. Bryan's patient after a long history of peptic ulcer covering twenty years became greatly emaciated from persistent vomiting, very little food being retained for four months. During this period of privation he developed what appeared to be the typical dermatitis and stomatitis of pellagra. Operation revealed carcinoma of the stomach, which was resected and gas-

tro-enterostomy performed. Following operation vomiting disappeared. Nutrition improved and within two months both skin and mouth lesions had entirely disappeared. Since Bryan's case was placed on record there have been 10 cases of pellagra complicating obstructive gastro-intestinal lesions added to the literature. Bender, in Journal of the American Medical Association for April 25, 1925, reports 3 instances, and Nuzum, in the same journal for December 12, 1925, adds 2 more. In 2 of Bender's cases pellagra developed during the course of gastric carcinoma after the lesion had caused obstruction to the passage of food. In Bender's third case there was a large ulcer on the lesser curvature of the stomach with hour-glass deformity and no food retention. At operation the stomach was found so involved in adhesions that gastro-enterostomy was impossible. Jejunostomy was performed with the purpose of again attempting gastro-enterostomy after a period of complete gastric rest. About a month after jejunal feeding was instituted symptoms of pellagra developed. The pellagra disappeared after the patient was placed on a general diet following gastro-enterostomy. Previous to Nuzum's report all recorded cases had been association of pellagra with gastric disease. Nuzum recorded the development of pellagra in 2 patients having annular carcinoma of the terminal portion of the ileum. In each patient well-defined skin lesions were present, and a diagnosis of pellagra had been made by dermatologists. At necropsy marked obstruction was found in each instance. These cases of Nuzum's appear to be the first recorded pellagra associated with carcinoma of the small bowel.

O'Leary, in an interesting clinic on Secondary Types of Pellagra, in Volume 10, No. 3 of these Clinics, presented the histories of 5 patients who showed besides the classical signs and symptoms of pellagra also demonstrable disease of the gastro-intestinal tract which antedated the onset of pellagra. The first of these patients developed pellagra apparently as a result of constant vomiting due to a gastrojejunal ulcer and a malfunctioning gastro-enterostomy. The second patient because of pain from gastric carcinoma limited his diet to a quart of milk

daily for one month, when the dermatitis and glossitis of pellagra appeared. In Case III carcinoma of the descending colon existed as an antecedent to the pellagra. In Case IV the primary factor was a malfunctioning gastro-enterostomy with persistent vomiting and Case V developed on a basis of benign esophageal stricture.

In the case herewith reported there was unfortunately no autopsy obtainable whereby to demonstrate the exact extent of the carcinomatous bowel involvement. Pretty accurate localization of the growth and its extent was, however, secured by barium roentgenography three months before death. Transit of the ilium appeared to be normal, but in the cecum a large filling defect existed, and there appeared to be a constriction at its juncture with the ascending colon. We may assume consequently that the lesion was a carcinoma of the colon and that the pellagra associated therewith was the result of underalimentation from a long-continued ineffectual attempt to control diarrhea by a diet of cereal, toast, and tea. The institution of an appropriate dietary brought prompt improvement, so that within five weeks thereafter all pellagrous manifestations had entirely cleared.

The etiology of pellagra has not been worked out to the agreement of all authorities. Students of the disease are divided into two great camps. One of these groups believes that the cause of the disease is to be found in food deficiency with emphasis on a too low protein intake, while the other believes the disease to be due to some form of infection the nature of which is unknown. An excellent account of the evidence on both sides is given by Edward Jenner Wood in Oxford Medicine. The preponderance of evidence both experimental and therapeutic appears to support the dietetic theory. The case herewith reported and those briefly recounted from recent current literature give added weight to the food deficiency theory of this disease.

CLINIC OF DR. JACOB MEYER

MICHAEL REESE AND COOK COUNTY HOSPITALS

GASTRIC ULCER HEMORRHAGE AND ITS TREATMENT

IN the past months I have observed 10 cases of gastric hemorrhage at the Cook County and Michael Reese Hospitals. Of these 10 cases, 1 occurred in a questionable duodenal ulcer, another in postoperative gastrojejunal ulcer, another in Banti's disease; 2 cases were diagnosed duodenal ulcer, but which exploratory revealed no intragastric or duodenal pathology, and 4 were gastric ulcers proved as such by clinical means. I will relate to you the history of the first patient.

He is a young man, twenty-seven years of age, who previous to a week ago was perfectly well except for an occasional discomfort described as heartburn. This discomfort has been yresent for five to six years. It comes on immediately and during his meals. It is not a pain, is localized indefinitely over the midsternal region, lasts for one to two minutes, comes on once or twice during the week, and is relieved by soda or milk. He has not paid any particular attention to this discomfort and is inclined to minimize the importance of same.

Seven days previous to admission to the Michael Reese Hospital (service of Dr. Sol. Strouse) he accidentally noticed that his stools were black. He continued at work until December 13, 1926, when he complained of headache, and felt dizzy, and his co-workers observed that he was extremely pale. He went home and summoned a physician, who advised him to go to the hospital. His history is otherwise negative.

On admission, a pale, restless young man who looks rather ill. The examination reveals no definite findings except slight

tenderness in the epigastrium. The pulse is rapid and of good quality. His thirst is moderate. He has not vomited. A specimen of stool was obtained and this was tarry. The diagnosis rendered was probable bleeding duodenal ulcer. Laboratory studies were now made. The red count was 2,400,000; white count, 11,700; hemoglobin, 45 per cent. A stomach-tube was passed on the patient on admission in order to determine the degree of acidity present. It was 26 free, total 68. The following morning an Ewald meal showed a free acidity of 52, total 64.

One month later on simple ulcer management the morning aspiration revealed no free acid and a total of 10. At the end of one month gastric *x*-ray studies were made and the diagnosis was duodenal ulcer. The course of treatment pursued is essentially that outlined below.

Hematemesis and hemorrhage in ulcer is a variable feature in the symptomatology as illustrated by this case. It may or may not be present; it may inaugurate the symptomatology or terminate the picture. Single hemorrhages are seldom fatal; they tend to recur. It is generally believed that peptic digestion causes a disintegration of the thrombus formed at the time of the primary hemorrhage, and that this digestion is responsible for secondary hemorrhage. Dr. Sippy has stated that "unquestionably the peptic disintegration of the thrombus before it has become sufficiently vitalized to offer resistance to the digestive action of the gastric juice, is an exceedingly important factor in the causation of recurrent hemorrhages at short intervals." It is on this basis that the late Dr. Sippy recommended the administration every half-hour of 60 to 30 grams of calcium carbonate and calcined magnesia, and for the first ten to twelve hours. He believed that complete neutralization of acidity would prevent any disturbance of the clot. Only one or two observers have studied the acidity at the time of hemorrhage, and they report that there is a marked increase of acidity at the time of hemorrhage and immediately after. This they say accounts for the changes in the character of the vomited blood, which is seldom bright red, but brown, having been converted into hematin by the hydrochloric acid of the gastric juice. I have been able

to study the acidity in 3 cases at the time of hemorrhage. The acidity figures are as follows:

		Empty aspiration: Free acid.	Total acid.
Duodenal ulcer	Proved by *x*-ray and clinical story	26°	68
Gastric ulcer	Proved by *x*-ray and postmortem. Developed hemorrhage during treatment	14	32
	(Sippy method)	10	40
Gastric ulcer	Treatment with alkalies two years; appendectomy; then grave hemorrhage; then operation with exploratory—no ulcer found, but chronic gall-bladder.	20°	40°

It may be true that there is an adequate acidity to promote peptic disintegration of the blood-clot, and in the light of Dr. Sippy's idea might justify the necessity for complete neutralization of all acid. The question arises, however, how are we to explain cases of hemorrhage that occur during the active course of alkali management. The question is analogous to the repeated observation of pathologists who will find a healed ulcer approximately a penetrating ulcer.

In my own experience I have not found it necessary to use alkali in the treatment of acute or recurring hemorrhage. A simple routine suggested is the following:

Absolute rest in bed, implying a restriction of all bath-room privileges. Morphin sulphate, $\frac{1}{4}$ gr. every four hours. At times I give 1/100 gr. of atropin with this, but not in cases where thirst is great. Nothing is given by mouth for the first twenty-four to forty-eight hours depending on the severity of the case. Minted chipped ice is given or patient is allowed to suck a few fruit tablets. An ice coil may be placed on the abdomen, but not made too heavy. At the end of twenty-four hours I give 1 ounce of a mixture of equal parts of iced milk and cream. This is continued from 7 A. M. until 9 P. M. During this day morphin sulphate is also given. Depending on the condition of the patient the quantity of fluid is gradually increased, not to exceed 3 ounces at any one time. On the third day, especially if there is distention, it is advisable to administer a low soapsuds enema. One or 2 tablespoonsful of mineral oil are also helpful. On the

fourth or fifth day, during all of which time the stools have been examined, it may be advisable to start routine ulcer management. In general, I follow the plan of treatment as advocated by the late Dr. Sippy on the routine case of uncomplicated chronic ulcer. I use the following formulæ for the powders, finding it more practical:

Powder No. 1
- Magnesii oxidi (heavy) ..... gr. xv
- Sodii bicarbonatis ..... gr. xv

Powder No. 2
- Calcium carbonatis ..... gr. xv
- Bismuth subcarbonatis ..... gr. xv

In severe cases of hemorrhage I am not an advocate of various intravenous medications, such as gelatin, horse-serum, etc. I have seen patients improve without it as well as with it. I consider hypodermoclysis of intravenous blood as a valuable adjunct. Every case of hemorrhage is kept in bed for a period of twenty-one days. The stools are examined daily. The presence or absence of blood in the stools is taken as an index of the efficacy of management. Complete gastro-intestinal *x*-rays are made at the end of one month's treatment or sooner if patient's condition permits. Patients who have a story of recurring gastric hemorrhage are referred to surgery following the recovery from the acute hemorrhage.

ACUTE NEPHRITIS—TREATMENT

THIS patient is a young man, twenty-one years old, who entered my service at Cook County Hospital February 6, 1927, complaining of pain in the lumbar region, blurring of vision, swelling of the face. Ten days previous to admission he became suddenly sick with sore throat, chills, headache, after which he remained in bed for a day or two. Returning to work he experienced pain in the lumbar region, general indisposition, and swelling about the eyes and face, which has increased. In addition he began to have severe headaches and vomited several times. His previous history is not of interest.

Physical examination reveals a young individual who is pale and whose eyelids are quite swollen. The tonsils are hypertrophied and injected. The heart and lungs are normal. There are no other findings. Blood-pressure is 170 systolic and 116 diastolic. The urine on admission shows a specific gravity of 1016, albumin +++, blood ++. Microscopic examination reveals red blood-cells in abundance, many granular casts, and a few white cells.

You will readily perceive that this is a case of acute glomerular nephritis with edema, for we have the cardinal symptomatology of hematuria, increased blood-pressure, and edema. Blood chemistry on the day of admission showed non-protein nitrogen 54 mg., urea 70 mg., creatinin 3 mg., cholesterol 166 mg. The changes in blood chemistry are confirmatory of a severe acute nephritis. On discharge from the hospital March 5th blood-pressure was 126/84, albumin +, blood chemistry −, non-protein nitrogen 41, urea 47, creatinin 1.7.

It is my purpose today to outline for you the course which you may pursue in the treatment of such a case. In general, the basic principle of the present treatment of acute nephritis is rest of the inflamed organ. The first step is to confine the patient to bed, advising warmth and comfort. It is known that

cold, exposure to wet, a cold room is often the cause of the onset of an acute nephritis. General body comfort is therefore the first essential. The second essential is rest of the kidney. Kidney function, as you know, depends in its greatest measure on the circulation through the glomeruli. It is believed by Volhard that in acute nephritis there is a vasomotor spasm or ischemia of the vas afferens, and that the changes in acute nephritis are a result of such ischemia. He therefore advocates what is now generally known as the "hunger and thirst cure of Volhard." This method consists in the restriction of all liquids and foods for a period of three to five days. During this time the patient may be given a cathartic, such as Epsom salts or castor oil. Also simple fruits, raw or cooked, or fruit juices in moderate quantities are given. Such a régime is, on the whole, not a great burden to the average patient. In a young child there is no particular difficulty. One is rewarded by the drop in blood-pressure, a gradual disappearance of edema, and an increase in the urinary output. At one time Volhard suggested that this period of hunger and thirst be followed by the administration of a great amount, 2500 or more c.c., of fluids, and in some cases a copious elimination is obtained. It is probably better not to use this procedure in most cases, but wiser to limit the fluid intake to the fluid output. The dangers of an excessive fluid intake are that it tends to throw excessive work not only on an already insulted kidney, but may overtax the circulation, producing cardiac failure.

We now come to the problem of the diet in acute nephritis. If we are to give our kidney rest, it is advisable to have a diet which will require least work of the kidney. Water, salts, and protein are eliminated with difficulty. The diet then should be poor in these substances and at the same time have sufficient caloric value to maintain weight. The general notion among students is that such a diet is largely made up of milk. It is well to remember, however, that milk contains a good proportion of protein, salt, and water, and that it is wrong and unwise to place a patient on a diet consisting of 2000 c.c. or more of milk.

It is well then to follow the first days of starvation, with the

continued administration of fruits, and fruit juices, with a sufficient amount of lactose and glucose. The sugars will aid the elimination by way of the gastro-intestinal tract, in that fermentation will be enhanced and diarrhea result. Foods, such as rice, cream of wheat, cream, and salt-free butter, potatoes, cauliflower, salt-free bread, butter, coffee, cocoa are added. The term "low protein salt-poor diet" is now generally used by a great many individuals who are not thoroughly aware of its meaning. It must be remembered that the caloric value of such a diet may not meet the caloric needs of the patient. This will not be harmful in the acute stage, but as the patient progresses and improves it is necessary to increase the caloric value of the diet. Cream, butter fat, and glucose are valuable foods for raising the caloric value in those acute cases which are very poorly nourished.

The fluid intake is often a source of great concern in the treatment of acute nephritis, particularly if edema is present. Without entering into a discussion of the different types of nephritis, it is probably best to limit the fluid intake to the fluid output in acute nephritis. The danger of embarrassing the circulation has already been pointed out.

Can we eliminate the toxins by forcing fluids? This is particularly the question on those cases of acute nephritis which develop anuria and uremia. On the whole, it would seem that the more reliable procedures in such cases are venesection and catharsis. It is probably well at this point to digress from the dietetic treatment to emphasize the complications of acute nephritis, for the therapy of these complications are most important in an understanding of the therapy of nephritis as a whole. Uremia and its accompanying anuria is best treated, according to Volhard, by the preliminary hunger and thirst cure, and many other clinicians agree that this is the simplest and best procedure not only in the prophylaxis, but in actual treatment of uremia. Venesection has already been mentioned. The amount of blood removed will depend on the blood-pressure and the condition of the heart. Removal of 500 to 800 c.c. of blood suffices in the usual case. Catharsis by the use of drugs,

such as magnesium sulphate, croton oil, elaterium, compound powder jalap are helpful. Sweats, by the dry heat or the wet pack, are often helpful, but it is important to watch the circulation.

It is also probably well to emphasize the very much neglected fact that every acute nephritic should be considered as a cardiac case. An acute nephritis, as you know, is associated not only with blood-pressure elevation, but with increase in pulse rate, some dyspnea, and, as the case progresses, with signs of circulatory embarrassment. Acute cases of nephritis may present the picture of an acute cardiac failure. This is rare, but it probably emphasizes the point that digitalis in the acute severe cases may be of great value.

One of the older conceptions of uremia was that it was due to a cerebral edema, and the withdrawal of spinal fluid by spinal puncture was therefore suggested as a therapeutic measure. In some cases striking relief is obtainable by this procedure.

Decapsulation of the kidney is sometimes advocated by some authorities in severe cases of acute nephritis with anuria. On the whole, I think you consider this measure as a last resort. The effect is often questionable and the explanation of the possible effects somewhat in doubt. At one time it was considered that the decapsulation relieves intrarenal pressure or spasm. Recently some writers, including Volhard, believe that the results are due to the non-specific protein reaction associated with the surgical procedure and entirely independent of the kidney decapsulation.

Returning now to the dietetic management of a case of acute glomerular nephritis, such as I have presented to you: as the edema subsides and the blood-pressure falls, and the urine becomes clear, containing only small amount of albumin, what procedures shall be followed? The diet can be gradually increased so that the patient receives not only a greater amount of carbohydrate and fat but also protein is increased to 40 to 50 grams per day. This amount permits a liberal choice of protein, and may include eggs and meat. There is a much

mistaken notion as to the dangers in meat and eggs. Well-cooked meats, from which all the substances have been extracted, probably are not as dangerous as the fried meats or roast or rare meat. Such meat as liver, thymus, kidney and brain, ham and sausages, and all spiced meats and spices are best avoided. As for eggs, I see no particular harm in including them in the diet if the total protein intake is restricted to about 50 grams per day. Vegetables are very desirable adjuncts in a nephritic menu. Among these may be mentioned asparagus, cauliflower, carrots, peas, turnips, squash, beets, string beans, etc. Your patients will also inquire as to spinach, rhubarb, and tomato, but personally I see no harm in them, even though they are known as oxalate-producing vegetables. Patients are to be advised as to the nature of the diet on discharge, recommending foods as indicated above, and advised to return in a period of three months for blood-pressure and urine examinations. During this period all foci of infection, such as infected sinuses, diseased tonsils, are to be treated and, if possible, the focus removed. The presence of an infected tonsil is a constant menace to an individual who has had an acute nephritis.

You will have observed that thus far I have not mentioned drugs other than the simple laxatives in the early stages, and the drastic cathartics in the stage of uremia. On the whole, all are fairly well agreed that drugs are of little value in acute nephritis. I might emphasize, however, that digitalis should be used in cases with early symptoms of cardiac failure.

CLINIC OF DR. JAMES G. CARR

COOK COUNTY HOSPITAL

AMEBIC DYSENTERY

THERE is a prevalent impression among the members of the profession that amebic dysentery is a rare disease in this part of the world. On the contrary, the disease is far from uncommon. We have an opportunity to demonstrate to you this morning 4 cases from our own ward. Such a number of cases in a single ward at one time is, indeed, unusual; on the other hand, such a group of cases is significant evidence of the frequency of the disease in Chicago. The records of the hospital show that during the past two years there have been 42 different persons in the hospital with amebic dysentery. Six of these were admitted twice, so that the files contain 48 charts. The increase in the number of cases in this section is, to a great extent, a real increase; the disease has occurred more frequently here in recent years. But the number of cases diagnosed is partly due to more careful study of the patients with diarrhea, especially of the chronic type, and greater hesitation about accepting the diagnosis of colitis of non-specific origin. Perhaps the close contacts of the Great War, here at home of men from all sections, and abroad of men from all parts of the world, provided opportunities for the spread of the infection. The increasing frequency of the disease and its more wide-spread distribution make it important that you should be familiar with the diagnosis and treatment of the disease. It is of first importance that you should realize that amebic dysentery must be considered as a possibility in the differential diagnosis of diarrhea. In the diagnosis of chronic diarrhea the careful examination of the stools for the Entamœba histolytica not once only, but many

times, is essential. The undiagnosed cases are apt to be the ones in which the possibility of amebic dysentery has not been considered.

The first patient is a Spaniard, twenty-six years of age, who has been in the hospital for more than five months. By occupation he is a cook, which fact carries its own implications as to possibilities for spreading infection. Upon admission he was complaining of a bloody diarrhea with cramp-like pain in the abdomen. He had been sick about six weeks; at the beginning of his illness he had about five or six stools a day; the condition grew steadily worse until, upon admission, he was having an almost constant desire to defecate and some two or three stools an hour. After he came under our observation there was a time during which he had thirty or more stools every twenty-four hours. The stools were quite bloody. His other symptoms were headache, progressive weakness, and loss of weight. There was no history of any previous illness except an uncomplicated appendectomy a few years ago.

Upon admission the temperature was 99° F., pulse 92, and respirations 20. At that time the record was made that "the patient is a well-nourished adult." As you may observe, he is now pale and emaciated. There was abdominal tenderness along the course of the large bowel. The hemoglobin was 65 per cent., the red blood-corpuscles numbered 4,175,000 per cmm., and the leukocytes 9200. The urine was negative. The stools were positive for ameba; both motile and encysted forms were found. An attempt was made to demonstrate the Shiga-Flexner bacillus, but the culture was negative.

A course of emetin hydrochlorid was given, with prompt response to the treatment. The period of improvement, however, was followed by a relapse in which the symptoms were more serious than they had been at entrance. A second course of emetin with opium and koalin was given, with almost no favorable effect, and for about a month the patient was in serious condition. The stools numbered thirty to forty a day, and often contained nothing but pure blood. The frequent defecations and the pain caused much loss of sleep; the patient was in a gen-

erally pitiable condition and his life was almost despaired of. There was a daily range of fever up to about 101° F. Stovarsol was tried after the emetin had failed to ameliorate the symptoms, but without any beneficial result. Later Chaparro amargoso was used, and under this treatment improvement began again. The convalescence has been very slow and throughout the summer the patient was confined to his bed continually. After some time, the amebæ being constantly present in the stools, a third course of emetin was given, while the Chaparro amargoso was given without interruption for quite a while. Nevertheless, the patient continued to have some six or eight stools a day, which contained blood and mucus. There was constant abdominal pain, increased at the time of a bowel movement, and the amebæ could almost invariably be demonstrated; only occasionally did examination fail to reveal their presence. After some three months with only this relative improvement, quinin irrigations (1 : 1000) were given twice daily for about three weeks. The effect of the irrigations was markedly beneficial. The number of stools decreased, the blood diminished greatly, and often stools are examined in which no amebæ can be found. The disease is now more nearly under control than at any time since his entrance to the hospital.

The second patient is a laborer of fifty-six, of Irish birth, who has lived in Chicago for twenty-eight years. He was admitted to the hospital five weeks ago. For seven weeks he had suffered with abdominal pain and diarrhea which he said had started while he was a member of an excavating crew in a down-state city. He reports that about half of his fellow workmen developed similar symptoms, which were of short duration in most cases. He has lost 25 pounds, and has had some headache, some frequency of urination, and pains in the legs. During the last week the stools have been blood tinged. He admits both gonorrhea and chancre, the latter about thirty years ago.

This patient is a well-developed white male. The temperature was 96.6° F., pulse 100, and respirations 24. About the umbilicus there was tenderness and increased peristaltic

sounds. Hemorrhoids were present. Upon rectal examination blood was found. Otherwise the general physical examination was negative except for a perforation of the hard and soft palate. The urine was negative. The hemoglobin was 80 per cent., the red blood-cells 4,200,000, and the whites 7600. The Wassermann was negative. Examination of the stools disclosed the presence of the Entamœba histolytica and of blood. This patient has had an uneventful course since coming under our observation. The temperature has not been above normal, the pulse has varied between 75 and 85. The diarrhea and pain disappeared within a week, but amebæ continued to be found in the stools. He was given emetin hydrochlorid, $\frac{1}{3}$ grain, twice a day for twenty-two days. He is now taking the Chaparro amargoso and daily irrigations with quinin solution (1 : 1000) are being given. During the first few days of his stay in the hospital he was given paregoric, bismuth subnitrate, and powdered chalk.

The third patient is a colored man, twenty-five years of age, a laborer by occupation. He has resided in Chicago about eighteen months. For six months he has had diarrhea, abdominal pain, and pain in the legs. The abdominal pain was quite severe, cramp-like in character, and limited to the lower abdomen. Recently he had been having about eighteen watery bowel movements a day. Some stools were bloody. He also complained of occasional dizziness, numbness of the hands, night-sweats, loss of weight, and failure of the appetite. His past history is negative except that he has had gonorrhea and smallpox. There is no history of a previous diarrhea. He smokes moderately, uses coffee, and occasionally alcoholic drinks. He formerly had the cathartic habit.

Physical examination revealed tenderness along the course of the large bowel and unusual tenderness of the rectum. Otherwise the general examination was negative. The Wassermann reaction was 2+ positive, the leukocytes numbered 9400. Blood and actively motile amebæ were found in the stools. Since his entrance into the hospital there have been only occasional slight rises of temperature and the pulse has been within normal

limits. Within a week after admission he was having only two stools a day. After four negative stools upon successive days he was permitted to go home. Three days later he returned with an attack of diarrhea, but after three days of observation, during which time no amebæ were found, he was again discharged. The diarrhea stopped promptly and was attributed to some dietary indiscretion. His treatment consisted of paregoric, tincture of belladonna, and bismuth subnitrate given during the first few days and of emetin hydrochlorid which was given in doses of $\frac{1}{3}$ grain twice a day for two weeks, and Chaparro amargoso, which was given for nine days during the course of emetin.

Note.—Three weeks after the second discharge this patient was readmitted with recurrence of the diarrhea and abdominal pain. Amebæ were again found in the stools.

The fourth patient, a man of forty-six, a laborer, born in Poland, and having lived in Chicago for sixteen years, came to the hospital complaining of diarrhea and abdominal pain of eighteen months' duration. The pain was described as dull and aching, occasionally becoming sharp. It was usually confined to the left lower quadrant. The pain had not been affected by the taking of food or medicine. Shortly before his admission the diarrhea had been more severe, as many as twenty stools having been passed in a day. The stools were dark and watery with streaks of blood. He dated the onset of his symptoms to an automobile accident in which he had sustained some injury to the abdomen not long before the onset of his diarrhea. About three months ago his physician called his attention to an abdominal tumor in the midepigastrium; this was tender to pressure. The appetite remained fair. There was occasional numbness of the hands and feet. His previous history included attacks of rheumatism, measles, pertussis, and influenza. He denied venereal disease. He has used tobacco and occasionally liquor.

At the time of admission his temperature was 98° F., pulse 88, and respirations 18. The blood-pressure was 118/60. In the midepigastrium there was a palpable mass about 3 inches in diameter, which was slightly tender. The tumor is hard and

moves with respiration. We regard it as located in the left lobe of the liver. The urine is normal. The blood Wassermann is negative. The hemoglobin is 80 per cent. and the leukocytes number 8400. The differential count is normal. The Ewald test-meal gave a free acidity of 10 and a total of 17. Blood and amebæ, motile and encysted, were found in the stools. The report of a roentgenologic examination of the gastro-intestinal tract stated that the findings were normal for the stomach, duodenum, and colon. The blood urea was 60.04 mgm. per 100 c.c. of blood and the blood-sugar 70.

Since this patient's admission his temperature has occasionally gone as high as 100° F. and the pulse has varied from 80 to 90. During the first month of his residence here he improved steadily. The diarrhea ceased and finally treatment was stopped. At this time the stools did not show gross blood; the chemical tests were not continued. After a few days without treatment the diarrhea recurred and amebæ were again found in the stools. The response to a second course of treatment was prompt.

In these 4 cases there is a common history of diarrhea and pain. In all the diagnosis of amebic dysentery was made and confirmed by the repeated demonstration of the amebæ in the stools. Only 1 of these 4 patients had come to Chicago from our own South. This man was in Chicago about one year before his symptoms began. A second one of the four, born in Spain, spoke English so poorly that we knew little about his places of residence except that he had been in this city about two years. Of the other two, one had been a resident of Chicago for twenty-eight years and the other for sixteen. Of the four, therefore, two have not lived in regions were amebic dysentery is regarded as endemic.

The diagnosis of amebic dysentery is not often difficult; if the possibility of the disease is kept in mind and careful examinations are made of fresh stools the amebæ are not often missed. Occasionally in older cases the amebæ are present in large numbers in the intestinal wall, but are missed in the feces. Such a case was reported a few years ago by Hirsch and Mayers

from St. Luke's Hospital. Although the diagnosis of amebic dysentery was considered, failure to demonstrate amebæ led to the diagnosis of ulcerative colitis of unknown origin. The patient succumbed and amebæ were found in the wall of the intestine.

Emphasis must be laid upon the examination of the stools while they are fresh and warm. Where stools are sent to the laboratory and allowed to stand for a while the reports may be constantly negative, even though the first stool properly examined may show amebæ in abundance. We deceive ourselves if we trust to routine methods for the diagnosis of this type of dysentery. Unless we make sure that the laboratory investigations are made with fresh stools, the results are of no value. A satisfactory substitute for the warm stool is the passage of the rectal tube, which, upon withdrawal, may carry enough mucus adherent to the tube or in the eye to permit the ready demonstration of amebæ. The experience which we noted with our third patient upon his return to the hospital for three days emphasizes the importance of repeated examinations of suspected, cases especially of those in which amebæ have been found formerly. A few examinations of the stools were made at this time and no parasites were found, upon which the man was discharged. Three weeks later, as the note states, he was readmitted with the old complaints of diarrhea and abdominal pain, and this time the amebæ were quickly found. A single examination of the stool is not diagnostic. Conclusions based upon a single negative report are far more apt to mislead than to help. In some cases with a history of chronic diarrhea which are seen at a time when the diarrhea is not present, a moderate dose of magnesium sulphate may be given; in the mucus which is passed with the resultant stool the amebæ may be found.

Recently a great deal of attention has been devoted to the subject of non-specific ulcerative colitis. Many of these reports have been based upon careful work and are valuable contributions to our knowledge. Yet this very discussion may easily lead many to leave an amebic dysentery undiagnosed. The truth is, that the amebic type of dysentery is far more prevalent

than is generally believed, and many cases of true amebic dysentery are undiagnosed because after a few rather careless examinations the conclusion is drawn that amebic dysentery is not common in these regions, and the failure to find the amebæ warrants the diagnosis of the non-specific type of the disease. We see many cases of dysentery at the County Hospital. Most of those coming under our observation have been due to malignant conditions of the bowel, to tuberculosis, or to amebic infection; some occur which are the result of advanced failure of the digestive processes, atrophy of intestinal mucous membrane, or pancreatic disease. The cases which have been diagnosed "non-specific ulcerative colitis" have usually been proved ultimately to be due to amebic infection. A chronic diarrhea of unknown etiology, in the course of which amebæ have not been demonstrated, should not be classed in the non-specific group because of the failure to find amebæ until the patient has been given a course of emetin without response to the therapy. In the latent or chronic stages of the disease the amebæ may have invaded the muscular coat of the intestine, as in the case already cited, or the ulcer may be deep and undermined, so that the parasites are not easily carried on with the intestinal contents. With an exacerbation of the diarrhea the increased secretion of mucus and the marked peristalsis will again carry out numbers of amebæ. The pathogenic organism is the Entamœba histolytica, which is characterized by the hyaline ectoplasm; often these parasites contain red blood-corpuscles.

None of the cases presented here had high fever. The first patient had a temperature of 101° F. while the symptoms were most severe. Usually the chronic case has little or no fever; with increased severity of symptoms the temperature may rise, but it is not high except in the acute cases; these are rarely seen in our wards. The appetite may remain fairly good, in spite of which the patient loses weight and strength. In 3 of our 4 cases the red blood-corpuscles numbered over 4,000,000 at entrance. All of these patients had been sick a month or more before coming under our observation. In the fourth case no count of the red blood-cells is recorded. Our first patient

became obviously anemic with the persistence of the disease and the constant loss of blood, but no second blood-count was made. The leukocyte count was slightly above normal in 3 of the 4 cases. This is in keeping with the usual findings in amebic dysentery; the leukocytic response to this infection is not marked.

One feature of interest is the duration of the disease as stated by the patients at entrance. The length of time during which symptoms had been present varied from six weeks to eighteen months. Only the last patient gave a history of having seen a physician, though it is not unlikely that the others had received at least occasional medical advice. However that may be, these patients came under our care after the disease was well established. None of them could be regarded as early; in all the pathologic changes in the intestine had progressed to such an extent that the time was past at which conditions for prompt response to treatment are present. It is probably true that many cases, diagnosed early and promptly given the specific treatment, will improve rapidly and often permanently. In the cases which have become chronic, as these had, every form of treatment may be followed by disappointing results. In such cases as these the symptoms recede slowly and tend to recur. While we have at our disposal several drugs which are regarded as specific, and which presumably will effect a prompt cure, experience teaches us that a "cure" in amebic dysentery is difficult. In these 4 cases there was not one in which the stools became negative for amebæ on three successive days in the first month after admission. One patient still has amebæ in the stools after five months. Of the other three, the stools of one are still positive after five weeks, and of another after seven weeks. If a cure is to be effected the disappearance of symptoms must not be interpreted as marking the termination of the infection of the bowel. Amebæ, both motile and encysted, may still be present. The carrier of amebæ is probably one great source of infection, though he may never have had symptoms or may have been long free from them. Failure to recognize that the infection long outlasts the period of symptoms is responsible for

many cases of chronic dysentery, though it may not be asserted that proper treatment, carried out until the stools are free of the amebæ for a given length of time, is certain to result in permanent freedom from relapse. In a patient known to have had amebic dysentery any attack of diarrhea should be regarded with suspicion.

In view of what has been said it is evident that prognosis in these cases is difficult. The prognosis as to life is almost invariably good, as we see the disease here, but the prognosis as to recovery and return to the usual occupation is a matter of doubt in any given case. At this hospital amebic dysentery is not often the cause of death. Where the diagnosis is promptly made and treatment instituted early the outlook for a permanent cure is fairly good. Where, as in these cases, the disease has lasted for weeks before specific therapy is used, remission of symptoms is often followed by a fresh outbreak. The absence of amebæ from the stools for some time gives no assurance of a permanent cure. Dietary indiscretions, exposure to cold or heat, mental or physical overwork may precipitate a fresh attack. The late results of the disease include the chronic type with prolonged course, emaciation and profound anemia, amebic abscesses of the liver, and occasionally stricture or perforation of the bowel.

The fourth case presents a problem in diagnosis. In the diagnosis of intestinal disease it is recognized that a persistent diarrhea may be due to malignancy of the bowel, especially in an individual of middle life or beyond. In this case a diarrhea existed of eighteen months' standing. Some fifteen months after the onset of the diarrhea a mass was discovered in the epigastrium. After the patient entered the hospital the diagnosis of amebic dysentery was made upon the discovery of amebæ in the stools. Specific treatment was followed by prompt disappearance of the symptoms. After a few weeks of freedom from symptoms, during which time the stools were occasionally negative for amebæ, another outbreak of symptoms occurred which was readily controlled by specific therapy. During the remission, in an endeavor to determine the nature and origin

of the abdominal tumor, the patient was examined roentgenologically. The colon was reported as negative. We believed that the mass described was a carcinomatous mass in the liver, though the primary disease could not be determined.

Note.—Exploratory operation showed a primary carcinoma of the sigmoid with secondary metastases in the liver.

The treatment of amebic dysentery may be discussed as palliative and specific. The palliative treatment includes the general measures employed in the management of acute infectious disease, the administration of liquid nourishment in the acute stage, followed by the addition of soft food as the symptoms subside and the use of hot applications to the abdomen. There is no unanimity as to the propriety of prescribing drugs for control of the diarrhea. In general, it is agreed that the opium preparations are indicated in the cases with profuse diarrhea, severe abdominal pain, and distressing tenesmus. The difference of opinion centers about the use of bismuth, kaolin, and intestinal astringents of all kinds. Many hold that these coat the ulcers and interfere with the local effect of any specific drug. As you have noted, we have employed those drugs during the first days of treatment. At this time the specific drug used was emetin, and the objection mentioned cannot apply to a specific given hypodermically. The continued use of such astringents is probably unwise; indeed, I am not sure that they are of value in this type of dysentery. Although we have employed them in an endeavor to control acute symptoms, it is my opinion that opium will give the desired results. We are likely to use more care with the administration of opium alone and to obtain better results because this is given with a definite purpose in view. The use of many drugs for the diarrhea, too, often means a careless use of all of these.

The specific treatment includes first of all the preparations of ipecac, which have become established all over the world. Emetin has been used in all these cases. This was given in doses of $\frac{1}{3}$ grain, twice a day, for twelve to sixteen days. In one instance this preparation was continued for twenty-two days; since it is usually accepted that about 12 grains of emetin

may be given in a single course, the amount given in the twenty-two days was about the maximum allowable for one course. We have not observed toxic effects in any of these men. Emetin is capable of producing toxic symptoms which are distressing; these include depression, cardiac weakness, and, most trying of all, a stubborn peripheral neuritis. The single dose should not exceed 1 grain a day and the maximum dose for a course should not exceed 15 grains. At least one week should intervene before a second course is given, and after the second course emetin had better be discontinued for some time. Another course given shortly after the second course is not unlikely to precipitate a neuritis.

We have found the Chaparro amargoso a valuable aid in the treatment of amebic dysentery. This was first described and recommended by P. I. Nixon, and has recently been advocated by A. A. Goldsmith, of Chicago, who believes, upon the basis of a large experience, that it possesses much therapeutic value. Chaparro amargoso is the name of a bush which grows in southwestern Texas and Mexico. An infusion is used in treatment following the directions of Nixon as quoted by Goldsmith: "In making the infusion the entire plant—roots, leaves, branches, and berries—are boiled from thirty to sixty minutes. No definite amount of drug for any given amount of water can be mentioned because the weight of the plant varies, depending upon its dryness. The color of the infusion should be about that of moderately weak tea." About 4 to 8 ounces of this infusion may be given by mouth a little while before each meal and enemata of 500 c.c. or more, dependent upon the amount the patient will tolerate, are given twice daily. While we do not believe that the Chaparro is as effective as emetin in producing permanent results, it has been our experience that no drug gives results more quickly. Indeed, we feel that the symptoms are apt to be more promptly ameliorated by the Chaparro than by any of the specifics.

Of recent years stovarsol has been highly recommended. While it has proved effective in some cases, it has been our experience that this drug is less dependable than the two already

mentioned. It may be that further use will bring more satisfactory results. At all events, it is fortunate that we have several specifics for amebic dysentery, for any one or all of them too often fail to effect a cure. Failing with one, another may be tried. Stovarsol may produce toxic symptoms and should be given carefully. In private practice I saw last fall a case of gastro-enteritis, associated with a dermatitis and followed shortly by a peripheral neuritis, in a patient who had been under treatment with stovarsol. The diagnosis of arsenic poisoning seemed well warranted. Emetin-bismuth iodid we have not used.

Finally, we must not neglect to emphasize that in at least one of our cases the quinin irrigations were of undoubted benefit. We believe this method of treatment is worthy of trial in the chronic cases especially. Irrigations of any sort are to be used with great caution in acute amebic dysentery. The pathologic changes in this type of disease are such that the bowel may be easily ruptured by instrumentation or even by overdistention with fluid. In the stubborn, refractory cases, however, the old-fashioned quinin irrigations ought to be tried.

CLINIC OF DR. JESSE R. GERSTLEY

DEPARTMENT OF PEDIATRICS, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL AND MICHAEL REESE HOSPITAL

OUR BABY PATIENT

LET me demonstrate a strictly modern baby. Judging from appearances this child is about one year of age. Notice his rosy cheeks and the clearness and freshness of his face. The delicate elastic skin is like velvet in its texture. Picking it up between the fingers one grasps no superficial wrinkles, but the well-developed subcutaneous tissue beneath. Feel the firmness of these leg and arm muscles. The tone of the abdominal muscles holds them down to the level of the thorax. There is no evidence of rickets in the bones of the head, thorax, or extremities. On the left upper arm is a vaccination scar and on the right a tiny mark like a pin prick with redness and induration beneath, the site of recent inoculation with toxin-antitoxin mixture. He holds his head up (a reaction of three months). He sits up (a reaction of six months) and when we support him under the arms he makes well-directed efforts at standing and walking. He has 6 teeth.

There is no question as to his mentality. He follows with his eyes and with his head, and he is not a bit bashful about reaching for bright objects.

This baby represents what one might call a strictly modern baby. His care includes not only the latest ideas of infant feeding but also the latest developments in the prevention of contagious disease.

First, let us discuss the feeding side of the situation. Some fifteen years ago during my internship a children's specialist was delighted with any method of artificial feeding that permitted a baby to live. He was not concerned with any of the fine points

of technic, but was satisfied if at the end of a year his little patient was alive. The attitude of the physician and the laity toward the artificially fed child was radically different from our scheme of today. This was due to the fact that medical science had not related many clinical syndromes to his artificial feeding. In those days a physician would prescribe a formula, and an even more important prescription, and then not see his little patient for weeks or even months. An emergency call would reveal the little one suffering from extreme malnutrition or severe diarrheal disease. I well remember my intern days. Night after night during the hot summer months I would be called from bed to administer first aid to newly admitted infants moribund from neglect. The treatment was hypodermoclysis. This revived the little one for one to two days. Not realizing the close connection between symptomatology and diet, the physicians were more content with the hypodermoclysis than correcting the underlying causes. The baby might live for two or three days and then would succumb. Hospital wards were full of feeding cases and the mortality was astonishingly high.

At this time an organization was founded called The Infant Welfare Society, the slogan being "Keep the well baby well." Organizations for reducing infant mortality have existed in many different cities and for many years, and their procedure has been based upon the scientific ideas then prevalent. In none, however, as far as I know, was there the simplicity in organization and function of our own Infant Welfare Society. Let me add here that the method of feeding employed in our Society was based primarily upon the studies of the great Europeans, Czerny and Finkelstein. Czerny's studies laid the foundation of a system of feeding which Finkelstein later reorganized and greatly elaborated. The system of feeding finally developed by Finkelstein consists of the use of milk mixtures, one-third, one-half, or two-thirds milk, with the addition of carbohydrates. Whatever success attended the infant welfare movement is due to the painstaking scientific study of these medical pioneers.

The Infant Welfare Society functions as a central bureau with stations scattered throughout the poorer districts of the city. The individual station is in some old store or possibly a park community house and is startling in its simplicity. There is a chair and an inexpensive examining table. In another corner is a scale for weighing the baby and chairs around the wall for the mothers. A well-trained nurse is present during the conference, helps the physician in his examinations, listens to the formula which is prescribed, and then follows the patient home, instructing her as to the actual procedure of preparing the bottle. Recently the Society has extended its activities to prenatal care as well as that of the preschool child.

The success of this novel experiment dramatic beyond description was striking in two ways. In the first place it was soon found that babies in these stations never developed the severe symptomatology that was seen in infants in the hospital. These children were well at birth and were kept well from the nutritional point of view as long as they came to the station. But something even more surprising developed; infants with almost identical symptomatology might be seen at the stations and in hospital wards. Upon identical formulæ the infants attending the stations would soon recover and thrive. At the hospital their illness became a long drawn-out affair and frequently ended fatally.

This experience was so uniform that it became apparent that the Infant Welfare idea had made a profound contribution to the study of infant nutrition. It showed that severe nutritional disturbance in infancy was not unavoidable, but an absolutely preventable condition. It showed the fundamental importance of home care in the life of the infant. The success of the Society was due largely to the nurse who went into the home, and who not only gave instruction as to the feeding formulæ themselves but also corrected all the hygienic defects that were present. She showed the mother how to keep the milk cool in summer, how to dress the baby, how to put the baby in the coolest part of the house in hot weather, how to clothe the baby properly in winter, how to ventilate the sleeping rooms,

how to screen for flies, and how to give the baby exercise. It soon became apparent that all these factors were as important in the baby's life or even more important than the food formula itself. In a home enlightened by such conscientious instruction and with a mother's care and devotion a baby was infinitely better off than lying like a plant in the hothouse of one of our well-equipped children's wards.

At this time the practitioner of pediatrics was in a peculiar situation. He saw his charity patients well nourished, thriving, and happy. This he had learned was due not to a formula alone, but to the home care of the infant; to the mother's improved knowledge of hygiene. His private patients who called him only when a child was sick were often, from the changing point of view, absolutely neglected. It was not long before the intelligent laity, particularly those who had had access to the work of the Society, began to demand such a service for their own babies. The life of the pediatrician was then drudgery indeed. The tyranny of the telephone permitted only an occasional house visit. The physician gave away gratis his priceless treasure, good advice, in hours spent at the telephone, and sold only the dregs, his medicaments. At this time some of us began to apply the idea of the Infant Welfare Society to our private practice. The results were so outstanding that I believe all pediatricians are unanimous in following our lead and practising in the same fashion. However, in the smaller communities the idea has not as yet taken root, and it is for that reason that I am dwelling upon it once more. If you wish to have little trouble with the feeding of your infants, if you want that phase of your work to be not a hardship, but really a pleasure, then apply this same procedure to your private patients.

See the baby as soon as it is born. Instruct the mother that it is far better to keep that child well, that it will be far less expensive to her to let you see the baby at certain intervals and keep it well, than to wait until the child is nine-tenths dead from malnutrition and then have you work with it for six weeks to save it from the grave. The procedure in all cases must vary with the locality and the clientele. In my own practice I make

a fixed charge for seeing the baby once a month for the first year. During this time I expect the mother to stay in touch with me over the telephone about once a week, so that the diet may be constantly adjusted to the immediate needs of the infant and so that any developing disturbance may be immediately checked.

In this discussion you notice I have said little of breast feeding. It is unnecessary to state that the baby should by all means be kept on the breast if possible. There will be little trouble with a breast-fed baby, and if the mother is in moderate circumstances a breast-fed baby need not be seen as frequently as a bottle baby. However, disturbances do occur in breast-fed babies, especially from insufficient breast milk, and for this reason by no means dismiss the breast baby from your mind, but give it also a considerable amount of attention.

Now let us turn to methods of immunization. About five years ago I was prescribing feedings for the child of a personal friend. He was thriving beautifully and at the age of about one year was a perfectly lovely baby. One day I was called in because the child had a slight cold. He did not appear very sick, had a temperature of 100° F., and showed no symptoms other than a slight coryza. For two days he remained this way. It was during an epidemic of influenza, and for that reason I told the parents to be particularly careful. On the third day the child developed a slight hoarseness and croupy cough. There was absolutely nothing in the throat and the culture was negative for diphtheria. During that epidemic a great many children showed these croupy symptoms which were probably of influenzal origin. Some had died with symptoms of laryngeal stenosis. Postmortem had shown nothing but influenza. However, to play safe, I gave the child a prophylactic dose of antitoxin. The next day the croup was somewhat worse. To be sure there was no exudate in the larynx I called in a prominent laryngologist. He used the laryngoscope, got a good view of the larynx, and informed the parents and myself that there was no sign of anything in the larynx. He and I left together, agreeing to come back in a few hours merely for

the sake of satisfying the parents. When I returned, whistling as I walked up the stairs in a care-free way, imagine my utter astonishment and chagrin at being told that half an hour after we left the child had dropped over dead. This was my first bitter experience in practice. Postmortem examination was refused. To this day I have no idea whether the child had an influenza and died of heart failure from the strain of the laryngoscopic examination, or whether the child may have had a diphtheria deep down below the larynx. I wished I had given more antitoxin. I have never quite gotten over that experience just because the parents were warm personal friends. One thing is certain, if that child at six months had had T. and A. inoculations I would have known then and would be at peace in my own mind with the knowledge that he did not have diphtheria.

This brings me to the subject of preventive inoculation. Let us limit ourselves to those which have proved of practical value. Just one word for pertussis. Whether the vaccine is of use or not is still an open question. I myself have never been convinced of its efficacy, so I should rather say little about it.

One word about typhoid. The period of immunization as we saw it in the army is limited to one year. Overseas we saw a number of cases occurring in soldiers who had been inoculated one and one-half years previously. I just mention that in passing to those of you who are interested.

Smallpox vaccination is of such proved value that we will devote little time to it. Smallpox was one of the great plagues of the Middle Ages. In some countries it caused one-half of all deaths. It was so common that it was exceptional for any one in a populous district to be free. Every child who survived, by the age of seven had pock marks. An old proverb stated "From love and smallpox no one remains free." In the sixteenth century it destroyed 3,500,000 people in Mexico; it nearly depopulated Greenland and Iceland. Of 12,000,000 deaths occurring in American Indians, 6,000,000 were due to smallpox. It destroyed ten times as many people as plague; in Europe alone nearly half a million people died annually, the majority of

these being children. Vaccination was introduced into the United States in 1799 by Dr. Waterhouse of Boston, some years following Jenner's publication, and it is interesting to note that the President of the United States, Thomas Jefferson, personally vaccinated some 200 people in that city. Following him Benjamin Rush took up the practice in Philadelphia, and then the campaign grew rapidly to spread the practice among the Indians and in the southern states where the negroes were proving so susceptible.

There is no age at which a child is immune, smallpox occurring in children of two weeks. Therefore do not hesitate to vaccinate children certainly not much after the sixth month. The immunity in many cases lasts for ten years.

The value of toxin-antitoxin mixture in immunizing against diphtheria is established beyond doubt. I know of no unquestioned bad result from the treatment. However, where three inoculations were considered sufficient some years ago, now we know some children require four or five. In my own practice I usually give three at intervals of one week. About a month later I make a Schick test, and if this is positive, give another one or two inoculations. The immunity established lasts a number of years. The amount of serum in the mixture is so small that it practically never produces serum sickness and it does not seem to interfere with the giving of serum if necessary later on in life. The Schick test, as you know, consists of injecting one-tenth of the diluted toxin *into* the skin. If at the end of twenty-four or, better, forty-eight hours there is an area of redness 1 cm. in diameter or more, and especially if there is induration, the test is positive.

The scarlet fever situation is exceedingly interesting. Without doubt the disease is caused by a hemolytic streptococcus which locates in the throat and sends its toxin throughout the body. The disease is, therefore, in every way analogous to diphtheria. There is an antitoxin for scarlet fever just as there is for diphtheria. It is put up in doses for prophylactic use and for actual treatment. This viewpoint, which is the one taken by the Dicks of Chicago, seems to be the one which has with-

stood all criticism. The antitoxin is prepared by injecting toxin into animals just as diphtheria antitoxin is made.

One word about the Dochez serum. This serum was made by injecting not the streptococcus toxin, but whole streptococci into animals and is, therefore, bactericidal, but such a serum has not stood the test of time and experiment. Observers state that whatever value it has is due not to its bactericidal properties, but to its antitoxin content, and its antitoxin content is naturally not as high as serum prepared by immunizing animals to large doses of toxin. As I understand it, all the sera on the market, no matter how they are labeled in packages, are antitoxins. Of the firms who have submitted their sera to standardization by the scarlet fever board, there are as yet only three—Squibbs, Parke Davis, and Mulford.

It was found that injecting 2/10 c.c. of diluted toxin into the skin of susceptible individuals gave a reaction of the same nature as the Schick test. This reaction must be read somewhat more rigidly than the Schick. It must be seen in twenty-four hours and if the area of erythema be over $\frac{1}{2}$ cm. is considered positive.

It has been found that by injecting susceptible individuals with five increasing doses of toxin an immunity to scarlet fever will be established which lasts a minimum of a year and probably considerably longer. Recently it has been found advisable to increase the number of inoculations to six, but I understand that within a short time the dose of each vial will be so increased that the five-inoculation method will be the one employed. It is a cumbersome method. The children sometimes react a little more severely than to the toxin-antitoxin mixture, but I have never seen any child feel the effects over twenty-four hours with the exception of one. He developed joint pains and the treatment had to be stopped. As far as I know the only firm licensed at present to put up this toxin for immunizing purposes is Squibbs.

Another method of immunization is being widely advertised at present, namely, the Larsen method. Claims are made that this mixture is effective after only one inoculation. I have had no practical experience with this latter technic, but have consulted a number of scientists who are attempting a careful study

of the question. They tell me that these claims are exaggerated, and that as yet the only absolutely reliable procedure we have is that of the Dick inoculations.

The measles situation is changing. Ruth Tunnicliffe, of Chicago, has described a Streptococcus viridans, occurring usually as a diplococcus, which seems to be the cause of measles. She has been able to reproduce the disease by injecting the organism into animals. Recently Fehring, of Detroit, has isolated a toxin from this organism. Two sera have been prepared, one a bacteriolytic serum by Tunnicliffe, and the other an antitoxin by Fehring. The use of either one is as yet somewhat experimental, but there is some evidence to show that the Tunnicliffe serum is the more efficacious. Just as in the other diseases, observers are attempting skin tests to determine immunity. One has two means at his disposal—a vaccine by Tunnicliffe and a toxin by Fehring. Just as with the sera, it appears that the toxin is not quite as reliable a test as the vaccine. There is nothing definite as yet as regards immunization of children against measles. All that we have are the sera for prophylactic and actual treatment.

How do these advances affect the practitioner? In a way it looks ominous. At first thought one would be inclined to fear the growth of preventive medicine as an actual means of destroying the income of the physician, but this is superficial reasoning. There is also another phase of the matter not so practical, but more idealistic. Physicians at heart seem to have a few more ideals than their fellowmen. I believe physicians at heart are somewhat kinder and more tolerant than the average person in the community. Unquestionably this is due to the fact that they are in constant contact with suffering and sorrow; that they are in close proximity to the human side of things in life. It has always grated upon me to feel that I was earning my living at the expense of some one else's suffering. It has always left a bad taste in my mouth when the parents of some nice little baby meet me on the street with a question, "Is there much sickness among children at present?" If I reply, "No, there is not as much this season as last year," their faces brighten and they say, "Oh that's fine, isn't it," and then their faces quickly

change, and they add, "I suppose it is pretty hard on you doctors though," and I am happy to say that I can reply, "No, we do not object to it at all, because we modern pediatricians are earning our living by trying to keep the babies well. We would rather do that than see them sick."

The modern method of practice has solved both problems. One sees the baby at least once a month for six to twelve months for advice as to feeding. About this time he is vaccinated against smallpox. Shortly afterward he gets three inoculations against diphtheria. As infants are so generally susceptible, it is unnecessary to make a Schick test before the treatment. One to two months after the inoculations one makes such a test. If this is positive, other inoculations are given. If negative, the child is given a rest for a year or two, when another Schick test is made. In a like manner in early childhood I frequently recommend the scarlet fever immunization. As the duration of this immunity has not been definitely established, I do not advise it as unequivocably as the toxin-antitoxin mixture.

This method of practice certainly is a method of the future. It provides a satisfactory income for the physician, but this income comes not from the occasional unfortunate child and unfortunate parents, but from every single patient in one's practice. But better than affecting the problem of income it fulfils the ideals of the physician and it makes the parents happy. Seeing your little patients develop into healthy, happy children changes medical practice from drudgery and strain to a real pleasure. This new viewpoint elevates the practice of medicine to the position where it really belongs. It makes professional ideals compatible with professional welfare.

To return to this little fellow, since birth his diet has conformed to the standards established by modern science. A conscientious nurse has carried the light of hygiene into his home. He is immune to smallpox. He will not contract diphtheria. In a few years he will probably become protected against scarlet fever. Contrast him to the pock-marked, strangling, undernourished waif of past centuries. Truly in one respect the world is making progress.

CLINIC OF DR. EMMET B. BAY

PRESBYTERIAN HOSPITAL

UNUSUAL PROGNOSES IN HEART IRREGULARITIES

OCCASIONALLY among the cardiac arhythmias a case occurs which presents what may be termed a "paradoxical prognosis." By this is understood that the usual prognosis for a given heart irregularity apparently does not obtain in the instance at hand; in fact, quite the opposite. Obviously this relation between diagnosis and outlook represents a personal opinion, and as such is subject to error, especially where that opinion seems contrary to the accepted one. In the cases here considered an effort will be made to show that the logic of the decision conforms with, rather than contradicts, clinical experience which is the sole arbiter in the matter. Three types of arhythmia are represented in this group. No doubt others might be found which would illustrate the point under discussion.

Case I is that of a young man who was in active athletics in college at the time his heart trouble was discovered. He had had no symptoms whatsoever, being able to play football, basketball, and baseball without undue shortness of breath or palpitation. He was one of a large group of undergraduates on whom routine electrocardiographic tracings were taken. His tracing showed the characteristic curve for right bundle branch block. He was informed of this and became extremely apprehensive. Being fairly intelligent, he went to the medical library of his college and investigated various types of blocks. One month later he consulted Dr. James B. Herrick, to whom thanks are due for the privilege of showing this case. At this time he could

and did talk glibly enough of "arborization block" and "myocarditis" and was very much worried. He had lost his appetite and, as a consequence, had lost 10 pounds in weight during the month. He had practically withdrawn from all activities at school. When questioned closely he had had no childhood illnesses other than measles. He was not subject to frequent sore throats or head colds. He could not remember having had "growing pains." He neither smoked nor drank.

At this time examination showed him to be an unusually well-developed young man. No congenital abnormalities were observed in the face, fingers, or elsewhere. The teeth were apparently in good condition with very little dental work. The tonsils were small and not hyperemic or cryptic. The chest was normal in contour and expansion was good and equal on the two sides. The lung fields showed no abnormal areas of dulness and the breath sounds were everywhere normal. The heart borders by percussion were within normal limits, and on fluoroscopy the heart was seen to be of average size and normal in contour. The heart tones were loud and clear. There were no murmurs over the precordium. The pulse was of good strength; its rate was 78, as was that of the heart. The systolic blood-pressure was 124; the diastolic 78. There was no tenderness or rigidity in the abdomen nor were any masses palpated. Examination of the extremities was essentially negative. An electrocardiogram taken at this time showed normal "P" waves in all leads with a "P-R" interval of 0.08 second. In all leads the "QRS" was broad —0.24 seconds, slurred, and slightly notched. The "T" was of opposite sign to the greatest deflection of the "QRS" in all leads. The latter was upright in Lead I and downward in Lead III. In short, it was the typical picture which Lewis, Williams, Wiggers, Pardee, Fred Smith, and others have proved to be associated with bundle branch block.

Ordinarily the pathology existing in cases exhibiting such an interference in the intracardiac conducting mechanism is a generalized process. A diffuse myocardial and in some instances endocardial inflammatory or degenerative change is present which disturbs the bundle branch. Rarely local lesions, such as

small endocardial inflammations, tumors, or granulomas, have been reported which have happened to be so situated as to produce this phenomena. It is practically always associated clinically with symptoms referable to the heart. Here, however, it is present in an individual previously almost abnormally active and healthy in whom it was found quite by accident. Furthermore, except for the electrocardiogram there was no evidence in this patient of a diseased heart or, indeed, of any abnormality.

Is it not reasonable to think that in this instance the usual prognosis for such cases, based on the probable pathology present, is incorrect? Here the pathology may only be guessed. An unusual, probably congenital, alteration in the conducting mechanism is a possible explanation. It seems almost a surety that had it not been discovered he would have gone on indefinitely as before until some new and probably independent difficulty arose. He has gone on with no apparent change in his well being for two years. Even the apprehension, which was the most serious feature of his case clinically, is now largely a thing of the past.

Case II is that of a senior medical student twenty-five years of age. He had had in the past frequent head colds followed by bronchitis and scarlet fever as a child of nine. He smoked from 15 to 25 cigarettes each day and had been doing so for several years. Occasionally he had noticed a slight vertigo which he associated with the "times when he was smoking too much." He was convalescent about two weeks from what was apparently an acute laryngitis and bronchitis when his attack of palpitation occurred. The week before he had slept little and smoked much while preparing for an examination. He was stationed in the Presbyterian Hospital as a clinical clerk when he developed his heart irregularity. He felt much as usual early in the morning, but at about 11 A. M. his heart suddenly began to pound. An electrocardiographic tracing taken fifteen minutes later showed a complete absence of P waves, a slightly wavy baseline, but not the regular wave of auricular flutter.

The ventricular complexes were spaced at irregular intervals. The QRS was sharp and not slurred. There was no preponderance of either ventricle. It was a typical curve of auricular fibrillation.

Examination during the attack showed the patient to be fairly well developed, but somewhat obese. The skin was moist and warm, temperature 98.2° F. The tonsils were missing; the mucous membrane of the mouth was slightly hyperemic. No cervical adenopathy or thyroid enlargement was present. The chest was normal in contour; the respirations were 22 per minute. There were no dulness or râles in the lung. The left heart border was 10 cm. to the left of the midsternal line. The apex impulse was forceful and no thrill was felt over the precordium. The heart tones were a little faint, but no murmurs were made out. The heart and pulse were completely irregular, but there was no pulse deficit. The rate was about 105 to the minute. He began to feel less palpitation, and in three-quarters of an hour without any medication the heart had returned to its normal rhythm, with a rate of 86 per minute. An electrocardiogram at this time and the next day showed normal curves with no disturbance in rhythm. The P waves were of average height and the PR interval was 0.12 second. The ventricular waves were normal. Except for the regular rhythm of the heart examination was essentially the same as previously. As well as could be determined by percussion on a relatively fat patient, the heart did not change materially in size or shape during or after the attack. This was the first such attack he had ever experienced, and so far, a matter of three years, he has had no other. The only change made in his routine was to cut down by half the number of cigarettes used per day.

It is difficult to think of a purely "functional" auricular fibrillation. There may be some relatively minor organic change in the myocardium of this patient. Yet it would seem that this case offers a much better prognosis—if not a completely satisfactory one—than those usual cases of auricular fibrillation associated with valvular heart disease, especially mitral stenosis, arteriosclerosis, or hyperthyroidism.

Case III is that of a man sixty-two years of age. He had had pneumonia twice as a young adult and typhoid fever at the age of thirty-five. He had passed insurance examinations at thirty-two, thirty-seven, and forty, with no difficulty. He had experienced no symptoms referable to the cardiovascular system which warranted a consultation with a physician before his present illness began. He remembered on questioning, however, occasional consciousness of palpitation extending back ten or fifteen years. One physician, under whose care he had been two years previously for a minor ailment, an acute rhinitis, had said after an examination that his heart was somewhat irritable. It is not known whether this represented a so-called "irritable heart" or an occasional premature contraction which was also present later. Eight months before he was first seen he had experienced an attack beginning with dizziness and blurring of vision in which he had lost consciousness for two or three minutes. He was said to have become very pale during this attack. He recovered quickly and spontaneously and resumed his daily routine the following day. He had another similar attack the following month and two attacks on the same day a week before he was seen. He admitted a little dyspnea on climbing stairs.

Examination showed the patient to be a rather spare, elderly man. All of the teeth were missing. The tonsils were small and atrophic. There was no thyroid enlargement present. The chest was slightly enlarged anteroposteriorly. The lungs were resonant, almost hyperresonant, throughout and there were a few squeaky râles heard on both sides both front and back. The left heart border was 10.5 cm. to the left of the midsternal line and the right heart border was substernal. There was no broadening of dulness at the base of the heart. The heart tones were clear; the aortic second sound was accentuated, almost ringing, although the systolic blood-pressure was 136 and the diastolic 92. There were no murmurs present. The heart rate and pulse were regular and 48 to the minute. A complete heart-block with a slightly rapid ventricular rate seemed probable. An electrocardiogram, however, showed

normal P waves before each ventricular complex, no P waves not followed by ventricular beats and a uniform PR interval of 0.16 second. The tracing also showed a high, slightly slurred QRS in Lead I, with a downward, also slurred QRS in Lead III, a left ventricular preponderance, which was thought to be compatible with his age and probable arteriosclerosis. The slurred QRS and the inverted T in Lead I spoke for a degenerative process in the myocardium, also part of the picture of arteriosclerosis. Nothing in the clinical history suggested a real coronary thrombosis with myocardial infarction as the basis for this myocardial change, and it is not believed that such obtained. This picture represented a sinus bradycardia and not a complete heart-block. Another tracing taken one week later when the nature of his arhythmia was known, and made while the patient inhaled and exhaled deeply, showed variations in heart rate during the phases of respiration. His pulse before starting this latter procedure was 64. This variation with respiration seemed to confirm the sinus origin of his arhythmia, though a true sinus bradycardia need not always exhibit this phasic character. In any case the first tracing taken with normal respirations showed equally spaced beats, though the rate was only 48. At another time an electrocardiogram showed marked variation in rate, with no apparent relation to respiration. On this tracing one diastolic interval was 2.66 seconds long. Some of these long intervals were interrupted by ventricular extrasystoles. Curiously enough, the patient experienced no discomfort during this transient standstill. However, it is believed that this represents the probable mechanism behind his attacks of syncope.

Mackenzie and others have emphasized the importance of this relatively rare arhythmia. It is to be classed among the sinus arhythmias. As is well known to all, sinus arhythmia is so common in childhood as to almost be properly considered the normal. Certainly ordinary sinus arhythmia bears no grave prognosis. Here, however, in a patient who exhibits some organic heart damage and who has already experienced attacks of syncope it cannot be disregarded. In fact, it seems highly probable that it should be considered in much the same light

as Stokes-Adams syndrome in complete heart-block. Some future transient standstill may last too long and become permanent. Unfortunately the outcome in this case is not known.

The cases detailed above all have prognoses opposed to the usual ones for each diagnosis. They tend to be more or less medical curios, but they are not without value in discouraging dogma and encouraging the correlation of clinical and laboratory studies. Neither alone would have given an accurate idea of the outlook in any of the instances given.

CLINIC OF DRS. CHARLES LOUIS MIX AND JOHN F. GOLDEN

MERCY HOSPITAL

OBSTRUCTION OF THE DUODENUM BY BANDS OF ADHESIONS

THE patient, a man fifty-four years of age, first came to the office June 11, 1926. At that time he complained of continuous pain in his abdomen to the left of the midline, about $2\frac{1}{2}$ inches above and $1\frac{1}{2}$ inches to the left of the navel. He said that this area was not sore upon pressure and that the pain in it was not aggravated by anything he could do, neither by moving, coughing, taking exercise, stooping or playing golf, nor by constipation, diarrhea, or the taking of meals. The only thing that has influenced the pain favorably is an enema. On cross examination it was evident that the pain was of a twofold character. It was like the constant throbbing pain which is probably peritonitic and the rhythmic, contracting, wave-like, cramping pain that is undoubtedly due to unstriped muscle-fiber contraction. When he has this pain he has a desire to urinate, but micturition does not relieve him. The pain is not referred to the back, groin, scrotum, or testes, nor does he have nausea and vomiting with it. At times he has pain beneath the right rib.

He states that fourteen years ago when he was forty years of age he had stones in his gall-bladder. He was operated upon and the stones and shriveled gall-bladder, which is said to have been functionless, were removed. At that time he weighed 136 pounds. Since then he has gained in weight and despite the pain weighs 149 pounds.

He is bothered a great deal with nocturia, yet he thinks micturition relieves him. He gets up in the night on an average

of every hour or hour and a quarter and, of course, is greatly concerned about his troubled sleep. He is somewhat constipated and has to take mineral oil and occasional enemata, yet he has never noticed mucus in his bowel movements, and he is not particularly troubled with gas in the bowel, though at times he is bloated.

The pain is not worse at any particular time day or night. This is of importance because he gives a history of a sore when he was sixteen years of age not followed by secondaries. He had a positive Wassermann a long time ago and since then several negative ones. He has taken a good deal of neosalvarsan, perhaps fifty or sixty injections in all. Two years ago he went to the Mayo Clinic for his trouble and a spinal test, which was made, was negative. He subsequently visited the Presbyterian Hospital in this city and had a spinal test, which was also negative. Evidently his case has been looked upon at times as the gastric crises of a spinal lues and there is good reason for doing so because he has a marked pupillary inequality, his left pupil being larger than the right, and he has mydriatic pupils. His knee-jerks are, however, present and the pupils react both to light and accommodation.

June 10, 1926 he went out to play golf, and during the first 11 holes he suffered intensely from pain. The pain then went away and the remaining 7 holes were played without any inconvenience. After returning home the pain came on again and he still had it the following day when he came to the office. He has had this pain now off and on for six years.

Four years ago he was examined intensively and a diagnosis of obstruction was made. He was operated upon, without relief, and was told that no obstruction was found.

Diagnosis.—In the face of his history it is difficult to come to an accurate conclusion. Inasmuch as there is a definite relationship between micturition and his pain and since the location of the pain is $2\frac{1}{2}$ inches above and $1\frac{1}{2}$ inches to the left of the navel, it seemed wise to have an examination made by the *x*-ray of his left kidney and to have repeated urinalyses made.

The first urinalysis made on the day of his entrance showed

an occasional red cell. On the 30th there was no blood. On August 13th, September 14th, September 17th, and September 20th blood-cells were present. On October 5th, 18th, November 4th, 15th, and December 6th blood was absent. On December 14th, 22d, 28th, and on January 7, 1927 blood was again present in all specimens. On January 25th it was absent. On February 17th it was again present, but on March 3d, 11th, and 14th it was absent. These findings are naturally rather confusing because of the variation. Blood was never present in any amount and he has never been conscious of any hemorrhage. At the most we found only an occasional red blood-cell. We have found now and then occasional granular casts and traces of albumin, but there is no reason to believe that he is suffering from any acute disturbance of his kidney. It was ultimately determined that the red blood-cells came from his prostate gland.

A roentgenogram was made on June 11, 1926, and it showed no definite shadow in either kidney indicative of a calculus. The left kidney shadow seemed normal in size and position. The lower pole of the right kidney was approximately 1 inch below the level of the crest of the ilium. On making this examination a marked exostosis was found involving the sacro-iliac synchondrosis and the fourth and fifth lumbar vertebræ and reported by the *x*-ray laboratory. We, however, felt that this exostosis was not the cause of his pain. On September 20, 1926 we repeated the examination of the kidney and again found no shadows indicative of calculus. The right kidney shadow was apparently higher than in the roentgenogram taken June 11th. Again the exostosis was found, the shadow involving the articular facets of the fifth lumbar vertebra. We could not feel, however, in tracing his history from week to week that he was suffering from a stone in the kidney, and we definitely abandoned the idea.

During the winter he complained at times of pain beneath his right rib at the site of the gall-bladder operation. We never could find any localized disturbance at this point, nor did we feel that there was any evidence that he had had an ascending process from the gall-bladder into the hepatic ducts of the liver.

His color was not good, but he had never been icteric. We definitely decided also that he was not suffering from any luetic disturbance. His seizures were not at all like those of gastric crises. For example, he never vomited with the pains, and he would have the pains not in paroxysms of short duration or for a day or two, but he would have continuous pain. Notwithstanding his pupillary inequality, his mydriasis, and his history we could not feel that he would be benefitted by antiluetic treatment. We decided in the course of time, however, notwithstanding the negative reports in regard to his operation four years ago when a diagnosis of obstruction was made and not found at operation, to investigate his alimentary canal. We should have done this probably in the first place, and would have done so had it not been for the fact that we understood it had been done carefully and that he had been operated on for the condition and that nothing had been found wrong in the abdomen. Yet, following his operation four years ago, his condition has been entirely unchanged as to the pain and its character.

He went to the hospital for observation once more March 20, 1927. Fluoroscopic examination showed a slight enlargement of the aorta which we attributed to his lues. His heart, however, was normal in size and position. He had no arhythmia or inequality of the pulse and no signs of any luetic manifestations. His diaphragm presented a normal appearance, and since he had never had any temperature or signs of hepatic abscess we did not consider this in our differential diagnosis to any extent. His lungs were fairly clear, but he is subject to a chronic bronchitis, as are many of the citizens of this locality.

Fluoroscopic examination of the stomach by means of barium showed the emulsion passing through the esophagus freely. The stomach was orthotonic. The greater curvature reached almost to the crest of the ilium with the patient standing. This was important because it shows a mild degree of block at the pylorus. *Our experience is that a mild gastroptosis is very frequently due to a slight obstruction of the pylorus.* His peristaltic function was quite active. Very noticeable was the fact

that the pylorus did not close squarely to the tip because of a filling defect on the lesser curvature side, and more important still was the fact that the first portion of the duodenum showed a number of small filling defects. Yet upon pressure the barium could be forced into the duodenal cap which smoothed out considerably. In the region of the second and third portions of the duodenum a condition of reverse peristalsis was seen. The third part of the duodenum was considerably enlarged, giving the condition known as megaloduodenum, being a dilatation secondary to an obstruction of some sort which was near the ligament of Treitz. The caliber of the duodenum seemed to be somewhat larger throughout than is normal. During fluoroscopy the patient complained of no pain or distress. Plates were taken immediately following and showed nothing that has not already been mentioned except an elongated appendix containing barium for forty-eight hours. The five-hour plate showed the stomach completely empty, so that there was no real blockade at the pylorus. The twenty-four-hour plate showed a small amount of barium present in the ascending colon, with a good deal of barium in the appendix, which was very much longer than normal, being fully 5 inches in length.

Besides being examined as to his stomach on March 21st, he was given a barium enema. The emulsion entered the colon with considerable difficulty and did not pass across the transverse colon from the splenic flexure, probably because of adhesions which prevented the passage of the barium. A good deal of spasticity was noted in that portion of the colon which was first filled.

As a result of this examination we felt very certain that there was no active ulcer of the duodenum or of the stomach. His gall-bladder had been removed so that it could not be accused of causing trouble. The duodenum was dilated and showed unmistakable regurgitation of the barium. It would come down to the bottom of the descending part of the duodenum, which was much enlarged, and then it would be shot back toward the pylorus. It did this again and again, so that evidently there was much trouble in emptying of the duodenum.

We could not feel that the duodenum was free. Apparently it was bound by adhesions which prevented a normal peristalsis and normal passage of duodenal contents. They could not get out of the duodenum. The behavior of the duodenum seemed to account for his troublesome pain. He often would complain of getting his attacks in waves lasting from ten to twenty seconds or longer and occurring in groups of three or five attacks. Apparently he would have five successive contractions of the duodenum before it quieted down and gave him a moment's peace. The *continuous* pain was regarded as probably due to continuous irritation of the peritoneum. We therefore told him that in our opinion the only thing that could produce any lasting benefit would be removal of the adhesions which were binding down the pylorus and duodenum and incidentally of his appendix. He readily assented to the prospect of an operation because he felt that life was not worth living at the present cost.

Unfortunately he was the victim of a subacute bronchitis at about this time, and because of the frequent cough and the large amount of purulent yellowish expectoration it was impossible to do anything. We had to wait until he could be approximately cured of his bronchitis and until his blood-count became fairly normal. On April 25th he still showed 11,700 white cells and 74 per cent. polymorphonuclears. By May 2d, however, the polymorphonuclears had dropped to 66 per cent. The normal being 65 per cent., we felt that it would be safe to operate upon him. We were not anxious to incur the possibility of a pneumonia, and consequently delayed the operation from time to time until his condition was absolutely favorable. The operation was done on May 6, 1927 by Dr. John F. Golden. I have asked him to give an account of what he did at the operation, which now follows.

Dr. John F. Golden: Dr. Mix having made the positive diagnosis of chronic obstruction of the duodenum due to adhesions accompanied by a clear-cut old chronic appendicitis, it was deemed advisable to do the operations separately; the appendix to be taken out first, the wound closed; and second, to

make a separate incision for the exploration of the duodenum, the incisions necessary being one over the appendix, the other epigastric.

The appendix was removed through the first incision with the utmost rapidity in order to conserve the operating time necessary for the duodenal operation. Adhesions of the appendix, its thickened walls and distention proved conclusively the correctness of the diagnosis of chronic appendicitis.

After closing the incision over the appendix, the second incision was made in the midline, exposing a dense, solid mass of adhesions between stomach and duodenum, stomach and liver, duodenum and liver, and adhesions to the anterior abdominal wall, also involving the hepatic flexure of the colon.

In this situation we located the ligament of Treitz and found the pathology entirely between the ligament of Treitz and the pylorus of the stomach. We worked upward from the ligament of Treitz and downward from the pylorus of the stomach, carefully separating the adhesions involving the duodenum. We succeeded in doing this to the complete satisfaction of Dr. Mix who carefully viewed the operation at every step.

In separating the adhesions of the duodenum, the stomach, the liver, the hepatic flexure of the colon together with the adhesions of the anterior abdominal wall were freed. Fortunately we had in the vicinity plenty of good strong workable omentum. The patient's condition at this stage of the operation allowed us to take plenty of time and we were able to cover all of the raw surfaces with omentum, specially covering and interposing omentum on the raw surfaces of the duodenum, satisfying ourselves that the adhesions would not recur.

Dr. Mix: Immediately following the operation his condition was good. The appendix was removed very readily, scarcely more than ten minutes being needed for its removal. The adhesions, however, required much more time, so that he was on the operating table from the time the incision was made until the abdomen was completely closed a little less than one and one-half hours. When he got back to his room he was rather

nervous and uncomfortable and moaned a good deal because of pain. He urinated only a few drops at a time, without relief. In view of the fact that he had been subjected to so severe an operation and fearing the possible advent of acetonuria, we ordered four 500 c.c. proctoclysis injections of 5 per cent. glucose and 2 per cent. bicarbonate of soda. This gave him 100 grams of glucose and 400 grains of bicarbonate of soda in twenty-four hours. He retained practically all of it because we dripped it not to exceed 40 drops to the minute. We gave the proctoclysis at intervals of six hours, so that he would have three hours of proctoclysis and three hours of freedom, alternating for twenty-four hours. He passed a very good night, his pulse not rising above 78. On the morning of the first day following operation at 10 o'clock he had a weak spell and he became rather cyanotic. His pulse jumped to 128 and then to 132, but with digifolin quickly dropped to 98 and then to 90, and all that afternoon it ranged from 82 to 84. He has unfortunately coughed quite a good deal following the operation, but he did not develop any serious disturbance of his chest.

May 23, 1927: The patient is still in the hospital thoroughly convalescent, planning for a future of much more pleasure than he had in the past. His temperature and pulse have been normal for days and he is quite free from pain even on full diet. We regard him as cured and the patient himself believes that he is now restored to health.

Note.—No *x*-rays accompany this report because the plates made were not nearly so satisfactory as the fluoroscopic examination report. Indeed, from the plates alone one could not make a diagnosis.